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SURGICAL TREATMENT OF MENIERE'S DISEASE.*†

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Operations on the labyrinth are the oldest form of surgical treatment for the relief of vertigo in Ménière's disease. They were in practice long before the exact anatomical basis for the disease had become clearly known. Knapp (1871) was the first to suspect increased intralabyrinthine pressure as the cause of the symptoms of Ménière's disease. Following him, Cheatle (1897) recommended decompression of the perilymphatic space as possible treatment of the condition, a suggestion which was later carried out by Jenkins in 1911. Meanwhile, opening and destruction of the labyrinth was first practiced by Milligan and by Lake (both in 1904). By 1930 about 50 cases had been reported in which the vertigo was successfully treated in this way. The majority of the otologists, however, rejected destruction of the labyrinth, mainly because of the danger of postoperative complications and because of the lack of exact knowledge of the pathology of the condition. This viewpoint is clearly reflected in Uffenorde's (1925) discussion of the subject. In the handbook

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edited by Denker-Kahler (1928) the possibility of surgical treatment of Ménière's disease is not mentioned. It seems that the danger of these operations had been greatly overestimated by the contemporary authors; there were remarkably few postoperative complications, and not one death reported attributable to the operation.

Since chemotherapy and the use of antibiotics have remarkably reduced the suspected or actual dangers of opening a living- noninfected labyrinth, the interest in labyrinthine surgery was greatly increased. A great stimulus was also given by the clarification of the clinical and anatomical picture of the disease.

It was recognized gradually that among the diseases which have the symptom of peripheral vestibular or labyrinthine vertigo in common, one group stands out clearly by its well defined sypmtomatology. This group alone deserves the name of "Ménière's disease," whereas all the other conditions constitute the large group of cases presenting "Ménière's syndrome."

True Ménière's disease is briefly characterized by attacks of peripheral vestibular or labyrinthine vertigo, reduced hearing and, in most instances, tinnitus, without any active disease of the middle ear or evidence of any other disease. The tinnitus is low-pitched and roaring or high-pitched, ringing or hissing in character.

The attacks of vertigo are characteristically paroxysmal and occur usually when the hearing loss and the tinnitus have reached their maximum intensity; but sometimes the vertigo occurs after the hearing has begun to improve. The intervals between the attacks are of various length, during which the vestibular symptoms usually disappear completely and the hearing improves; but the tinnitus may persist, particularly if it is of the low-pitched variety. The condition lasts for many years, sometimes throughout life. The dizzy spells disappear eventually in many cases, but the hearing loss is progressive. The tinnitus usually persists longer than all other symptoms.

The functional status of the diseased vestibular apparatus shows fluctuations. It is lowered in the later stages of the disease and particularly during the attacks. In the intervals between the attacks, central regulatory processes compensate for the difference in the functional status of the two labyrinths. During the attacks the equilibrium is again upset; the attacks may recur as long as the diseased labyrinth shows even the slightest amount of function.

The threshold of hearing shows wide fluctuations, particularly in the lower and middle ranges. During the attacks the thresholds drops sharply but may go back to normal, particularly in the early stages of the disease. In more advanced cases the fluctuations become less marked, and the hearing loss does not return to normal or improve markedly in the intervals between the attacks. A permanent and progressive hearing loss develops which is frequently, but not always, more marked in the lower and middle ranges or extends more uniformly over the entire tonal range. In cases in which the disease starts in the middle age or later, a high tone loss may already be present at the time of the onset; however, this high tone loss is not related to the disease. In advanced cases the hearing loss may become very marked, but in rare instances only is it complete. In our experience, recruitment of loudness is frequently, but not always, present. A sense of blockage in the affected ear and intermittent diplacousis binauralis dysharmonica, a disturbance in which the same tone is heard at a different pitch in both ears, are frequent findings.

The clinical picture has a satisfactory explanation, through the findings in autopsy material. They show dilatation of certain parts of the endolymphatic labyrinth, a so-called endolymphatic hydrops. The changes in the end-organs are difficult to evaluate; it seems that the attacks cause a temporary compression of Corti's organ with transient and, to a certain extent, reversible changes in the hair cells; it is not improbable that similar changes, perhaps less evident, might also occur in the other end-organs.

According to Mygind and his school, not all the cases of Ménière's disease present the picture of endolymphatic hydrops. In many of them one must assume the presence of what he calls "endolymphatic compression" resulting from an overpressure in the perilymphatic system. This first stage might be followed by endolymphatic dilatation. Mygind quotes the histologically examined cases of Videbech and Arnvig as samples of Ménière's disease with endolymphatic compression. The case of Videbech, however, does not show the characteristic clinical picture of true Ménière's disease. It neither proves nor disproves Mygind's theories. The history in Arnvig's case is not detailed enough to establish beyond doubt the diagnosis of true Ménière's disease. If the endolymphatic dilatation is really the second stage of a process which begins with endolymphatic compression, it would be hard to understand why, in a patient with a clinical history dating back for 40 years and with repeated attacks of dizziness for the last 25 years, the histological changes should not have gone beyond the first stage.

As long as more convincing evidence to the contrary is not presented, one must regard endolymphatic hydrops as the anatomical basis of true Ménière's disease. It is, however, probable that in the early stages of the disease the endolymphatic system regains its original shape after the attacks. Later, the distension becomes permanent, although the endolymphatic pressure returns to normal. Mygind's opinion "that there is no borderline between Ménière's disease and, on one side, all the labyrinthine complications that may arise in connection with middle ear lesions and, on the other side, a number of endocranial affections with labyrinthine phenomena" is misleading and not confirmed by the histological findings in typical cases of Ménière's disease. The latter should rather be regarded as disease *sui generis*, unrelated to other conditions with a more or less similar symptomatology.

Unless this sharp distinction is maintained, it is very difficult to evaluate the efficacy of treatments used in this

condition. A proper evaluation is, however, of the greatest importance, because it is obvious that surgical treatment should be considered only after medical treatment has proven ineffectual.

Cases which clearly show the clinical picture of Ménière's disease are not too frequent in my experience. The very large series of cases supposedly observed by some authors within a relatively short period of time might be due to a less strict selection. Another point which is easily forgotten is that the untreated cases of Ménière's disease show a tendency to spontaneous remissions with return of the hearing to the previous level and, often, disappearance of the tinnitus within a few weeks; the remissions may last for many years. The attacks of dizziness eventually become weaker spontaneously, no matter what treatment is used and finally disappear completely.

Judging from my own experience and that of our department, I am unable to share the optimism of many authors concerning the chances for a permanently and completely successful medical treatment of fully developed cases of Ménières' disease except the rare bilateral cases where streptomycin achieves an incomplete or complete loss of function of the vestibular portions of both labyrinths. In the great majority of cases of true Ménière's disease followed in our department during the last years, surgical treatment has eventually been necessary after the usual medical treatments gave only temporary relief of the condition.

Operations are performed on the labyrinth, the VIIIth nerve, the autonomic nervous system and the cisterna pontocerebellaris.

Operations on the VIIIth nerve belong to the domain of the neurosurgeon and shall not be discussed in detail in this paper. It shall only be mentioned that complete section of the nerve relieves the vertigo but destroys the hearing. It is in its effects not superior to destruction of the labyrinth but is a more extensive and hazardous procedure. Differential section

of the vestibular portion attempts to preserve the hearing, but the results of the operation are often disappointing. In many instances the dizziness persists and the hearing becomes greatly reduced, due to the technical impossibility in many instances of cutting all the vestibular fibres and leaving the cochlear fibres intact. Section of the nerve, even in successful cases, does not influence the pathologic process in the inner ear; even if the vertigo is relieved, the hearing is bound to go down, due to the continuation of the attacks of endolymphatic hydrops.

The operations on the labyrinth consist of simple decompression of the perilymphatic space, complete or partial destruction of the membranous labyrinth or drainage of the endolymphatic space through incision of the endolymphatic sac.

The results of simple decompression of the perilymphatic space of the lateral semicircular canal (Jenkins, Hautant, Lindsay, 1949, and others) are unsatisfactory. In four cases of Lindsay, the opening in the bony wall of the canal stayed patent for more than a year. In three of them, a needle had been introduced through the window, and the nerve to the ampulla of the lateral canal had been destroyed. These patients were partially relieved from the dizzy spells. The hearing was reduced but not completely destroyed; the very unpleasant roaring noises persisted. Three of his patients showed a sensation of unsteadiness and nystagmus in the presence of loud noises, the so-called Tullio phenomenon.

A practically complete destruction of the labyrinth can be achieved through the classical methods of labyrinthectomy which might be combined with extraction of the stapes and the union of the round and oval windows into one large opening (Milligan, Lake, Yearsley, Matte, Jones, Botey, Hautant, Bourguet). Less extensive operations with a simple mastoidectomy as a preliminary step and closure of the retroauricular incision at the end of the operation attain the same results through opening of the vestibule from behind

and destruction of its contents with a small dental excavator (Goodyear), or through exposure of the membranous lateral semicircular canal and excision of a small piece of it (Cawthorne). Contrary to our expectations, the latter procedure leads invariably to a complete loss of labyrinthine function. This is difficult to understand because injury to the semicircular canal in the course of a fenestration operation frequently causes no further loss of hearing. All the aforementioned authors use the retroauricular approach. Lempert advocates an endaural transtympanic approach with preservation of the drum, extraction of the stapes, removal of the round window membrane and enlargement of the window itself. All these operations eliminate the attacks of vertigo but completely destroy the hearing.

Although the majority of cases unquestionably show unilateral involvement, estimates of the frequency of bilateral disease vary greatly. According to Day, it is around 10 per cent; according to Wright, 28 per cent. The latter reports that an additional 13 per cent of the originally unilateral cases later on showed an involvement of the other ear. The findings in the limited number of histologically examined cases seem to be more in line with the higher figures given by Wright. No matter what the final figures are, the possibility of a secondary involvement of the other apparently healthy ear must always be kept in mind. This fact limits considerably the usefulness of destructive operations.

The effect of the operations on the tinnitus varies. Matte, who first tried to analyze the character of tinnitus in Ménière's disease, found that the very annoying pounding and roaring noises disappeared after destruction of the labyrinth, whereas high-pitched, less unpleasant "seething" noise persisted; in the author's opinion, it probably originated in the nerve trunk. A similar differentiation between two kinds of tinnitus was more recently made by Day. In 20 cases operated by Milligan the tinnitus was completely relieved in eight, partially relieved in four, and unchanged in eight.

Lempert, without differentiating between the two kinds, believes the tinnitus to be most likely of cochlear origin. In his opinion, it disappears in those cases in which a complete and "atraumatic" degeneration of the cochlear part of the endolymphatic labyrinth, including the organ of Corti, takes place. Incomplete degeneration will not relieve the tinnitus. In case of degeneration as a result of postoperative infection, it will also continue, due to the cochlear neuritis caused by the extension of the inflammation from the organ of Corti to the cochlear nerve trunk. Lempert believes that such a complete atraumatic degeneration takes place in the manner he suggests. He reports disappearance of the vertigo in all 10 cases operated by him, with relief of the tinnitus in nine. Two cases in our department were recently operated after this method. In the first three months after the operation, the roaring noise was less intense but not relieved.

From the available material, it is impossible to draw any conclusions as to the possible seat of origin of the tinnitus and the causes for its disappearance or persistence. This is well illustrated by the fact that in a large series of cases in which labyrinthectomy had been performed for purulent labyrinthitis, tinnitus was strikingly rare, whereas in another series of cases in which healing had taken place without operation, tinnitus was often present (Schlander, Vienna, personal communication). On the other hand, tinnitus frequently persists after complete section of the VIIIth nerve, even though done under sterile precautions and without any evidence of postoperative infection.

Destruction of the labyrinthine contents has also been attempted by injection of dehydrated alcohol. This is done either through a hole in the lateral semicircular canal after opening of the mastoid (Mollison, Mill, McDowall) or through the stapes footplate into the vestibule (Wright, Peacock) or through the promontory into the basal cochlear turn (Berggren) without opening the mastoid. The imposibility of controlling the outflow of the injected alcohol from the labyrinth has in some instances caused facial paralysis. For this rea-

son this method seems less advisable than the open surgical approach.

A selective destruction of the vestibular part of the labyrinth through electrocoagulation of the contents of a semicircular canal was first attempted by Putnam. He made a small subtemporal decompression, elevated the dura as for the section of the trigeminal root, drilled a hole into the superior vertical canal and introduced a coagulation electrode into the lumen. He was able to relieve the vertigo and to preserve the hearing. The tinnitus remained unchanged and persisted even after secondary destruction of the cochlea.

Day enters the mastoid through a retroauricular incision, exposes and opens the lateral semicircular canal, introduces a fine coagulating electrode through the opening into the vestibule and coagulates with a weak current. His method has been widely accepted because of its relative simplicity and its good results. A great number of operations has been reported without serious postoperative complications (Williams, Ireland, Lindsay and others).

Fifteen cases in our department were operated upon by this method. Bone dust was put into the fistula after the coagulation in order to secure a bony closure. Penicillin was given during the entire postoperative stay in the hospital, which averaged 10 days. No serious postoperative complications attributable to infection occurred. A transient facial paralysis in one case was evidently due to the use of too strong a coagulation current and too large a needle. A similar observation was made in a case of McNally. Like Day, we also found that the postoperative labyrinthine disturbances were much less severe than one would expect after sudden destruction of the labyrinth. It is easy to understand that they were least marked in those patients who had had preoperatively a marked hypofunction of the labyrinth. The results of the operation, as far as the elimination of the dizziness is concerned, are uniformly good. The hearing is not invariably lost, as in the previously described operations, although it

might decrease to a varying extent. Among the 21 cases operated by Day, three retained usable hearing; in one of them, it improved from a 70 db. loss to normal hearing, which was maintained for four years.

In our 15 cases the vertigo was completely eliminated. The hearing was completely lost in 11. Among the remaining four cases, it became worse in three. The average additional postoperative hearing loss was 20 to 25 db. in two cases, 35 db. in another. Postoperative improvement averaging 15 db. was noted in one case. In none of these cases did the postoperative hearing loss stay above the 30 db. level. In one of them, the loss for 512 was 30 db., for 1024 and 2048, 40 db., 14 months after operation.

In a few of Day's cases, there was a transient postoperative improvement with eventual loss. From these findings, Day believes that an open perforation must have existed in the membranous labyrinth which caused a gradual loss of endolymph and a consequent loss of hearing. He also assumes that if the coagulating current could be so controlled as to seal off the perforation of the membranous organ made by the needle, the chances of preserving the hearing would be improved. A slight withdrawal of the needle at the first application of the current, followed by a second or third application with the needle thus withdrawn, would seem to offer a better chance of sealing off the perforation of the membranous organ. If the hearing loss is really due to damage to the organ of Corti in consequence of increased endolymphatic pressure, it would be difficult to understand how further endolymphatic pressure increases could be prevented after the perforation has been sealed off.

The low-pitched roaring noise disappeared completely in 19 of the 21 cases reported by Day; in two of them it remained to a very slight extent. A high-pitched tinnitus, however, remained and in some cases became even more pronounced. Among our 15 cases, the ringing disappeared completely in four and remained unchanged in five. In two cases it changed

to a continuous high-pitched ringing, and in four cases there were occasional attacks of slight, high-pitched ringing. In none of the cases was the postoperative tinnitus regarded by the patient as unbearable or even particularly annoying. There was no definite relationship between the postoperative behavior of the noise and the hearing condition. Among the cases in which the tinnitus completely disappeared, the hearing was either completely lost or, on the contrary, improved; in those cases in which it remained uninfluenced, the hearing was either decreased or completely lost. Where the tinnitus became high-pitched postoperatively, the hearing was lost in all but one case where it only decreased.

The analysis of our cases shows that complete relief from the dizziness and permanent as well as intermittent relief from the tinnitus is compatible with preservation of hearing.

The greatest drawback of Day's method, as practiced today, is the impossibility of an exact control of the extent of the coagulation. This explains the varying but usually unsatisfactory results obtained with this method with regard to the preservation of hearing.

Animal experiments carried out on monkeys fail to clarify the situation. Schall and Tom Rambo showed that with the use of an adequate coagulating current, the contents of the vestibule and the semicrcular canals were destroyed and the cavities filled with fibrous tissue which shows a marked tendency to ossification. Changes in the cochlea varied from absence of any damage to a serous and serofibrinous exudate filling both the scalae vestibuli and tympani. There was, however, no definite relationship between the amount of damage to the cochlea and the time elapsed since the operation or the amount of the coagulating current used. Slides demonstrated by Day of a human case with a long lasting postoperative hearing improvement which was maintained through several years showed the changes referable to the operation confined to the vestibule and the semicircular canals. Experiments in monkeys carried out by Lindsay consisted in coagulation of

the ampulla of the lateral canal, coagulation of all canals at maximum distance from the vestibule, injury to the nerves, to the horizontal canal ampulla and to the utricle, and use of very weak coagulating currents. In no case did the injury result in any extensive change or fibrosis in the perilymphatic space of the vestibule. The histologic findings failed to provide an explanation for the profound loss of hearing which occurred in humans in which the coagulation procedure had been limited to canals or their ampullae only.

The unpredictable effects of the coagulation upon the hearing limit its usefulness again mainly to cases without usable hearing in the affected ear; otherwise, coagulation should be used in exceptional cases only. As far as relief from vertigo and tinnitus is concerned, coagulation is as effective as completely destructive operations. The operations attempting partial destruction, however, hold much greater potentialities for further progress. It seems possible that eventually a method of intralabyrinthine surgery will be worked out which will preserve the hearing.

A different and theoreticaly sound approach to the problem was devised by Portmann. He believed that increased endolymphatic pressure was the cause of the Ménière attacks, an assumption which later was confirmed by the anatomical findings. Using a retroauricular transmastoidal approach, he exposes the dura in Trautmann's triangle and exposes and incises the sac which lies between two layers of the dura. By reducing the endolymphatic pressure, this operation is supposed to eliminate the attacks of dizziness and to restore as much as possible the original condition of the entire endolymphatic system. In early cases the hearing is expected to improve; in those of longer duration, it is expected to remain at the preoperative level. The cause for the attacks is not eliminated. but as long as the opening in the endolymphatic sac remains patent, the endolymphatic pressure will increase very little, if at all, during the attacks and cause only slight, if any, clinical manifestations.

The operation does not entail any particular dangers. In cases of sclerotic mastoids and marked forward and lateral position of the sinus, injury to the latter and bleeding might occur, which, however, is easily controlled by packing. In case of bleeding, the operation should be stopped and finished a few days later. Whereas in the previously described operations the operative cavity is allowed to fill up with blood in order to secure the formation of dense scar tissue, great care must be taken in this operation that no blood accumulates in the cavity, because organization of the coagulum might lead to rapid closure of the opening in the endolymphatic sac. Sometimes it is difficult or even impossible to be sure that the sac is actually opened. In these cases it is suggested by Woodman to lift the dura from the posterior surface of the petrous bone towards the internal auditory meatus. In this way, the endolymphatic sac is torn at the place where it leaves the bone and enters the dura.

Although the idea of the operation is theoretically sound, it has a very serious drawback in the inability to secure the permanent patency of the opening in the sac. The results obtained by me in eight cases are far less favorable than those reported by Woodman and by Waltner. Woodman, for instances, reported that among 11 operated cases the vertigo was cured in eight and relieved in one. The tinnitus was cured in two and improved in one. The hearing was improved in six and unchanged in two. Waltner reviewed Portmann's cases and found that the vertigo was always cured, the tinnitus improved in most of the cases and the hearing improved in many of them. The results obtained by other authors were less favorable. Parkinson found in one case, two years postoperative, that the vertigo was reduced to occasional mild feeling of unsteadiness for about one hour's duration. There was a decrease of the tinnitus, but a progressive loss of hearing. Cawthorne got unsatisfactory results in several cases. According to Harrison, the immediate results of the operation are excellent; later, a fibrosis around the saccus develops and the patient returns to his previous state or a gradual loss of function results which renders the operation pointless.

Among the eight cases operated by me, two have remained completely free from attacks; one so far for three years and one for 16 months.* In the former the hearing improved postoperatively, in the latter the hearing became slightly worse; the ringing decreased in intensity but did not disappear completely. In the third case the attacks disappeared for one year but came back, evidently due to involvement of the other ear. In the fourth case there were no attacks in the three months elapsed since the operation. In the fifth case the attacks started to recur after nine months and a coagulation of the labyrinth was performed. The other three cases were outright failures. In one of these a postoperative hemorrhage into the mastoid cavity had caused a rapid obliteration of the lumen of the sac and a coagulation of the labyrinth was successfully performed; in the two other cases the cause for failure was not so obvious. In one of these a complete section of the VIIIth nerve was done and the vertigo disappeared. The other patient, treated with streptomycin, showed improvement but not complete disappearance of the vertigo. This case, however, is difficult to evaluate, due to the presence of a marked psychoneurotic factor. The hearing was improved in two cases; unchanged in two; slightly worse in another two, and markedly so in still another two cases. The tinnitus disappeared completely in the two cases in which the hearing has improved, remained unchanged in two, and became reduced in intensity in the four others.

The results with Portmann's operation in this series are obviously not impressive; nevertheless, it seems worthwhile to try the operation in all cases with serviceable hearing in the affected ear, particularly when the hearing in the other ear is not normal. All our patients are informed about the limited chances of success from the operation and the reasons why it, nevertheless, should be tried. It is explained to them that if the intervention is unsuccessful, a coagulation of the diseased labyrinth could be done as a short second operation.

*Meanwhile the first of the two cases had to undergo a coagulation of the labyrinth.

Anatomical specimens of operated cases are unfortunately not yet available. The limited number of cases in which Portmann's operation brought unquestionable permanent relief from vertigo contradict Lindsay's statement that drainage could have only a limited influence on labyrinthine hydrops and that any effect of the operation could be of only relatively brief duration. Lindsay comes to his conclusions from the results of experiments on monkeys. He found there that simple drainage and various degrees of destruction of the saccus as well as destruction of nearly all of the endolymphatic duct were followed by prompt healing and filling out of the defect with newly formed bone. The remainder of the endolymphatic system and the sensory organs showed no histological variation from the normal. Ohma, however, in one case of a human with obliteration of the endolymphatic duct, found dilatation of the cochlear duct, the saccule and the utricle. The possibility should also be considered that in the presence of hydrops the chances for permanent patency might be better than in a normal endolymphatic system.

Operative interventions on the autonomic nervous system in Ménière's disease are performed on the assumption that labyrinthine hydrops might be the result of neurovascular and possibly neurosecretory changes in the labyrinth (see Hilger). The influence of the autonomic nervous system upon the labyrinth can be seen from the results of animal experiments and operations on humans. Constriction of the labyrinthine blood vessels causes an increase in the excitability of the vestibular portion; dilatation of the blood vessels leads to a diminution in the excitability of the vestibular portion and an increase in the hearing acuity (see Frazer). The operations which have so far been undertaken in Ménière's disease attempt to secure the inhibition of the vasoconstrictor impulses to the inner ear in order to secure a more stable flow of blood. Elimination of spasms of the labyrinthine vessels is supposed to prevent the development of the hydrops and in this way to eliminate further attacks. The interventions include decortication of the common carotids, excision

of the cervical sympathetic trunk and of the superior, middle and inferior cervical sympathetic ganglia, and various combinations of these procedures. The immediate results of these operations have been good, but in the majority of cases the symptoms have gradually returned within a few weeks or months, particularly in cases in which operations on the carotid arteries alone were performed. This becomes understandable in the light of Frazer's experiments in cats where the internal ear can be completely deprived of its vasoconstrictor impulses by cutting the cervical sympathetic trunk but not by decortication of the carotid arteries. Removal of the stellate ganglion or operations on the vertebral arteries are unnecessary in cats as long as the sympathetic trunk is interrupted above the ganglion.

Good results were lately reported after cervical ganglionectomy by Biancalana and by Mogan and Baumgartner. In the observation of the latter two authors there was no recurrence of the vertigo, no further deterioration of hearing and only occasionally slight tinnitus.

Garnett Passe and Seymour have recently reported a series of 12 cases of Ménière's disease in which the stellate ganglion was removed. In four of them, in addition to the stellectomy, the vertebral artery was either ligated or divided; in two cases it was decorticated. In 11 cases the vertigo attributable to the operated ear was completely relieved. Tinnitus was completely relieved in three cases and reduced in most of the others. The relief seemed more pronounced with the pulsating type of tinnitus than with the continuous variety. The perceptive type of deafness from which each patient had suffered showed improvement in most of the cases, with the exception of those in which the deafness was already very far progressed and beyond relief. Horner's syndrome was present after the operation. According to the authors, it rarely troubled the patients after the first few days and tended to correct itself within a few months. The diagnosis of true Ménière's disease was not established beyond doubt in all the cases and the time of postoperative follow-up was very short

in many of them. In some cases the symptoms could have been due to vascular (arteriorsclerotic) changes and elimination of vasoconstrictor impulses should have had a particular beneficial effect. The results in the cases with intervention on the vertebral artery were not better than in those without intervention. The assumption that constriction of the labyrinthine vessels plays an essential part in bringing about the Ménière attacks needs confirmation through further clinical and experimental research before it can be unreservedly accepted; but even now, interventions on the cervical sympathetic system should be regarded as a promising rational treatment not only of Ménière's disease but also of other inner ear conditions, particularly deafness of the perceptive type.

Drainage of the pontocerebellar cisterna is helpful in cases of Bárány's syndrome, which is supposed to be caused by increased tension within the cisterna. Bárány's syndrome, however, is not related to true Ménière's disease, in spite of a somewhat similar symptomatology. If interventions on the lateral cisterna are mentioned by some authors among the treatments for Ménière's disease, it shows again only the confusion which still exists with regard to the meaning of the term "Ménière's disease" and the necessity for a clear-cut definition of it.

SUMMARY AND CONCLUSIONS.

The term "Ménière's disease" should be confined strictly to cases which show all the classical signs and symptoms of the condition. It should not be applied to even slightly questionable cases, because this would confuse the issue and constitute a great danger for the proper evaluation of the results of any kind of treatment.

Ménière's disease or endolymphatic hydrops is a relatively rare disease with a known pathology but, so far, unknown etiology. It seems to be a disease *sui generis* and not related to other conditions with a similar symptomatology.

Contrary to the experience of other authors, we have found that fully developed cases of Ménière's disease can very rarely be permanently cured by conservative measures and that ultimately surgery or streptomycin therapy must be considered in most of them. This discrepancy is probably due to the very rigid standards used in our department for the selection of the cases and our insistence upon a follow-up over a prolonged period of time before we regard a case as cured.

For a proper evaluation of the results, one should distinguish between two groups of cases: those in which the vertiginous attacks have completely disappeared and those in which they have persisted. To create a special group of "improved cases" seems of questionable value because one has to rely too much upon the patient's evaluation of the severity.

In the evaluation of the tinnitus, special attention should be given to the character of the tinnitus and possible changes brought about by the operation. Differences in the intensity seem of lesser importance.

In spite of our great skepticism regarding the chances for success of the medical treatment, it should be given a fair trial in each case before surgery is considered.

Among the various kinds of surgical treatment, interventions on the autonomic nervous system seem promising, but have not yet progressed beyond the very first experimental stages.

Endocranial surgery achieves good and reliable results for the relief of vertigo in cases in which the nerve is completely cut; the hearing is completely lost and the tinnitus not seldom persisting. The same results are, however, achieved with less extensive and less hazardous operations on the labyrinth. The results of differential section of the vestibular nerve are sometimes gratifying but as a rule out of proportion with the magnitude of the intervention. It seems, therefore, that at the present time operations on the labyrinth are

a more rational way of surgical treatment of Ménière's disease.

Operations on the labyrinth can be divided into two groups: The first group tries to achieve either complete destruction of the labyrinth or localized destruction of the vestibular portion with preservation of the cochlear part; operations of the second group attempt drainage of the endolymphatic system with subsequent reduction of the endolymphatic pressure.

The completely destructive operations eliminate the dizziness together with all residual hearing, but their effect upon the tinnitus is less certain. Time alone will tell whether certain types of destructive operations are more successful in this respect than others. The usefulness of these operations is confined to cases with unilateral involvement and, perhaps with rare exceptions, to cases without usable hearing in the affected ear. To sacrifice hearing in the involved ear appears risky in view of a possible future affection of the other ear. For the same reason, operations in cases with bilateral involvement must be regarded as too dangerous, even if the symptoms point definitely to the more affected ear as the source of the actual troubles.

Operations aiming at selective destruction of the vestibular portion have been equally successful in the elimination of the vertiginous attacks. Although the hearing might be preserved or even improved in a small number of instances, it is impossible to tell beforehand when this will occur. For this reason, the indications for these operations remain essentially the same as for the completely destructive procedures. Coagulation of the membranous labyrinth, according to Day, is the most widely accepted procedure and has given satisfactory results in the 15 cases operated in our department.

The indications for drainage of the endolymphatic system (Portmann, Woodman) are much wider and comprise all the cases of Ménière's disease, including those with usable hearing. Judging from our experience with eight cases, the operations are successful in only a very limited number; neverthe-

less, the operation should be tried in cases where preservation of the residual hearing in the affected ear is essential. In case of failure, it can be followed by coagulation of the labyrinth.

At the present time, it cannot be foreseen whether the technique of intralabyrinthine surgery will eventually be so far improved as to achieve an isolated destruction of the vestibular portion in all cases. If not, the possibility still remains that operations on the autonomic nervous system will be the ultimate answer to the challenging problem.

BIBLIOGRAPHY.

BARANY, R.: Der Baranysche Symptomenkomplex, seine Diagnose und Therapie, Atiologie und Prognose. *Verhandl. dtsch. otol. Ges.*, 21:108, 1912.

BERGGREN, E.: Contribution Toward the Surgical Treatment of Morbus Ménière. *Acta Oto-Laryngol.*, 27:626, 1939.

BERGGREN, S.: Die Bedeutung von Golls und Burdachs Kernen bei Ménièreschen Anfällen. *Acta Oto-Laryngol.*, 15:234, 1931.

BIANCALANA, L.: La stellectomia per la cura delle sindromi di Ménière postototiche. *Ann. di laryngol.*, 39:41, 1939.

BOTEY, R.: Die Trepanation des Labyrinthes bei Schwindel und Ohrensausen. *Monatsschr. f. Ohrenh.*, 48:255, 1914.

BOURGUET, J.: Traitement chirurgical des vertiges labyrinthiques par l'evident des canaux semicirculares. Methode et technique personnel. *Bull. et Mem. Soc. de Chir. de Paris*, 22:175, 1930.

CAWTHORNE, T.: The Treatment of Ménière's Disease. *Jour. Laryngol. and Otol.*, 58:363, 1943.

CHEATLE, A. H.: Quoted after E. C. Jones.

DAY, K. M.: Labyrinth Surgery for Ménière's Disease. THE LARYNGOSCOPE, 53:617, 1943.

DAY, K. M.: Hydrops of Labyrinth (Ménière's Disease). Diagnosis. Results of Labyrinth Surgery. THE LARYNGOSCOPE, 56:33, 1946.

DAY, K. M.: Discussion to Lindsay, *Trans. Amer. Acad. Ophthal. and Otolaryngol.*, 53:534, 1948-1949.

FRAZER, P. F.: Vasomotor Control of the Ear. *Jour. Laryngol. and Otol.*, 51:579, 1936.

GOODYEAR, H. M.: Ménière's Disease. Surgical Approach to the Labyrinth with Destruction of the Utricle; Case Reports. THE LARYNGOSCOPE, 53:743, 1943.

HARRISON, M. S.: Ménière's Disease. *St. Thomas Hosp. Gaz.*, 43:88, 1945.

HAUTANT, A.: Traitement chirurgical du vertige auriculaire. *Ann. d. mal. d. l'oreille et d. larynx*, 45:924, 1926.

HILGER, J. A.: Autonomic Dysfunction in the Inner Ear. THE LARYNGOSCOPE, 59:1, 1949.

IRELAND, P. E.: The Diagnosis and Treatment of Ménière's Symptom Complex. *Canad. Med. Assn. Jour.*, 58:269, 1948.

JENKINS, G. J.: Labyrinthine Vertigo (Ménière's Symptoms — Non-infective) Treated by Operation. *Proc. Roy. Soc. Med.*, 4:116, 1911.

JONES, H. E.: The Operative Treatment of Aural Vertigo Due to Causes Other Than Suppuration. *Liverpool Med. Chir. Jour.*, 32:445, 1912.

KNAPP, H.: A Clinical Analysis of the Inflammatory Affections of the Inner Ear. *Arch. Ophthal.*, 2:204, 1871.

LAKE, R.: Removal of the Semicircular Canals in a Case of Unilateral Aural Vertigo. *Lancet*, 1:1567, 1904.

LAKE, R.: A Case of Operation on the Vestibule for the Relief of Vertigo. *Lancet*, 1:26, 1906.

LAKE, R.: Ten Cases of Operation for Ménière's Disease (Aural Vertigo). *Lancet*, 1:1569, 1911.

LAKE, R.: Aural Vertigo (Nonsuppurative): A Clinical and Therapeutical Study. *Lancet*, 2:1638, 1912.

LEMPERT, J.: Lempert Decompression Operation for Hydrops of the Endolymphatic Labyrinth in Ménière's Disease. *Arch. Otolaryngol.*, 47: 551, 1948.

LINDSAY, J. R.: Healing of Experimental Labyrinthine Fistulas. Further Observation. *Arch. Otolaryngol.*, 46:584, 1947.

LINDSAY, J. R.: Effect of Obliteration of the Endolymphatic Sac and Duct in the Monkey. *Arch. Otolaryngol.*, 45:1, 1947.

LINDSAY, J. R.: Labyrinthine Surgery for Ménière's Disease. THE LARYNGOSCOPE, 59:22, 1949.

MATTE: Zur Chirurgie des Ohrlabyrinthes. *Arch. f. Ohrenh.*, 73:142, 1907.

MATTE: Labyrinth-Operation wegen hochgradigsten Labyrinthschwindels. *Arch. f. Ohrenh.*, 86:243, 1911.

MCDOWALL, G. D.: Surgery of Ménière's Disease. *Jour. Laryngol. and Otol.*, 62:420, 1948.

MCNALLY, W. J.: Quoted after Lindsay, 1949.

MILL, W. A.: The Operative Treatment of Ménière's Syndrome. *St. Thom. Hosp. Rep.*, 1:192, 1936.

MILLIGAN, W.: Ménière's Disease. A Clinical and Experimental Inquiry. *Brit. Med. Jour.*, 2:1228, 1904.

MILLIGAN, W.: The Surgical Treatment of Suppurative and Certain Nonsuppurative Affections of the Labyrinth. *Jour. Laryngol. and Otol.*, 39:245, 1924.

MOGAN, R. F., and BAUMGARTNER, C. J.: Ménière's Disease. *Arch. Otolaryngol.*, 41:113, 1945.

MOLLISON, W. M.: Operative Treatment of Vertigo. *Proc. Roy. Soc. Med.*, 28:1597, 1935.

MYGIND, S. H.: Histologic Studies of the Labyrinth. XI—Correlation of Our Histological and Clinical Experiments and Observations. *Acta Otolaryngol.*, 35:108, 1947.

MYGIND, S. H.: Further Labyrinthine Studies. *Acta Oto-Laryngol.*, Supp. 68, 1948.

OHMA, M.: Uber einige Beziehungen krankhafter Veränderungen der Labyrinthliquor-Räume und Liquorkanäle zueinander. *Hals. usw. Arzt.*, 30:73, 1939.

PARKINSON, S. N.: Ménière's Symptom Complex; Endolymph Decompression with Symptomatic Improvement; Report of a Case. *Ann. Otol., Rhinol. and Laryngol.*, 44:382, 1935.

PASSE, E. R. G., and SEYMOUR, J. S.: Surgical Treatment of Ménière's Disease. *Brit. Med. Jour.*, 1:812, 1948.

PEACOCK, R.: Ménière's Syndrome. *Lancet*, 2:1409, 1938.

PORTMANN, G.: The Saccus Endolymphaticus and an Operation for Draining the Same for the Relief of Vertigo. *Jour. Laryngol.*, 42:809, 1927.

PUTNAM, T. J.: Treatment of Recurrent Vertigo by Subtemporal Destruction of the Labyrinth. *Arch. Otolaryngol.*, 27:161, 1938.

ROYLE, N. D.: Alteration of the Circulation of the Brain by Surgical Means in Diseases of the Central Nervous System. *Brit. Med. Jour.*, 1:1063, 1932.

SCHALL, L. A., and TOM RAMBO, J. H.: Electrocoagulation of the Membranous Labyrinth. Experimental Studies in the Monkey. *Ann. Otol., Rhinol. and Laryngol.*, 57:590, 1948.

TERRACOL, J.: Nystagmus et sympathique cervical. Recherches Experimentales. *Ann. mal. d. l'oreille, d. larynx d. nez et d. pharynx*, 46:1128, 1927.

UFFENORDE, W.: Die chirurgischen Erkrankungen des innern Ohres. *Hand. d. spez. Chir. d. Ohres u. d. ob. Luftwege*, hrsg. v. L. Katz and F. Blumenfeld, 2:657, C. Kabitzsch, Leipzig, 1925.

VIDEBECH, H.: Alteration du labyrinthe se presentant sous les traits du syndrome de Ménière. *Acta Otolaryngol.*, 22:51, 1935.

WALTNER, J.: Le blocage du sac endo-lymphatique et l'operation de Portmann. *Rev. d. Laryngol.*, 61:1, 1940.

WILLIAMS, H. L.: The Present Status of the Diagnosis and Treatment of Endolymphatic Hydrops (Ménière's Disease). *Ann. Otol., Rhinol. and Laryngol.*, 56:614, 1947.

WOODMAN, E. M.: The Position of the Portmann Operation in Relation to Vertigo. *Proc. Roy. Soc. Med.*, 32:1642, 1939.

WRIGHT, A. J.: Aural Vertigo: A Clinical Study. *Proc. Roy. Soc. Med.*, 31:87,1937.

WRIGHT, A. J.: Labyrinthine Destruction in the Treatment of Vertigo by Injection of Alcohol Through the Oval window. *Jour. Laryngol. and Otol.*, 53:594, 1938.

WRIGHT, A. J.: The Deafness of Ménière's Disease. *Jour. Laryngol. and Otol.*, 58:379, 1943.

YEARSLEY, M.: A Case of Severe Vertigo; Destruction of the Labyrinth; Cure. *Lancet*, 2:871, 1908.

YEARSLEY, M.: Treatment of Labyrinthine Vertigo by Operation. *Clin. Jour.*, 63:383, 1934.

II PANAMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

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THE USE OF THROMBIN AND ABSORBABLE SPONGE FOR HEMOSTASIS IN OTOLARYNGOLOGY.*

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In a previous study, the anatomy, histopathology, incidence, causes, and methods of control of nasal hemorrhage in a series of hospitalized cases were discussed.[1] The techniques and substances which were tried and discarded in the search for satisfactory methods of hemostasis were outlined. In the course of this study an opportunity was afforded to evaluate the efficacy of thrombin and absorbable sponge in the ear, nose, and throat.

A number of excellent reviews on the development of hemostatic agents have appeared. Great advances have been made in neurosurgery with the early use of electro-cautery, muscle implant, and the silver clip.[2] Later thrombin[3,4] and fibrin foam[5] were successfully isolated for control of capillary and venous bleeding. More recently the use of absorbable sponge,[6] absorbable cotton,[7] casein fibrin,[8] and starch sponge[9] have been suggested for surgical hemostasis.

There are some limitations to the usual techniques for the control of bleeding in various otolaryngologic procedures. During mastoid surgery it is undesirable to use coagulation current on the dura, on the lateral sinus, around the facial nerve, or in the middle ear. When performing operations on

*This study made possible by a grant from the Upjohn Company, Kalamazoo, Mich. Absorbable sponge is marketed under the commercial name of "Gelfoam."

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the nasal septum and sinuses, ligation of bleeding points is not usually possible, and cautery for the control of bleeding may not be practical.

Because the subject has been widely discussed, little need be said here about thrombin except that its discovery and development was a most important step in the problem of hemostasis. Seegars *et al.*[10] successfully used thrombin from beef plasma. Its low toxicity when applied locally was noteworthy. It has been shown by Tidrick *et al.*[11] that the use of thrombin provides an excellent surgical adjunct in the control of oozing from capillaries and small venules.

A water insoluble gelatin base sponge was prepared by Correl, Prentice, and Wise[6] which when implanted in tissues was almost entirely absorbed in thirty days with little tendency to encapsulate. Slight tissue reaction occurred and there was only moderate cellular infiltration. These investigators demonstrated that gelatin sponge implants become more gelatinous and transparent with time. Only a small film could be found on the twentieth day. Histologic identification of any unabsorbed material was doubtful after the twenty-fifth day. Slight cellular infiltration was noted maximally between ten to twenty-five days and then seemed to recede.

Gelatin sponge has been demonstrated to be a pliable substance when moistened and capable of absorbing many times its own weight of fluid, thus acting as an excellent carrier.[12] It has been shown to be non-antigenic and non-pyrogenic. It does not inactivate penicillin and can be readily used with thrombin without prior neutralization.[13] Jenkins and Clarke[14] reported that gelatin sponge had hemostatic properties of its own and used it as a supplement to ligature or where ligature was inappropriate.

Pilcher and Meacham[15] found that gelatin sponge was useful in the control of epidural venous bleeding, particularly under a bone edge, in diffuse tumor bed oozing, and in preparation of a dry bed for a sutured peripheral nerve. A survey by these authors of the results obtained by various neurosur-

geons revealed that the sponge was "satisfactory in the majority of cases."

Light and Prentice[16] emphasized the use of the sponge for the repair of torn venous sinuses and control of dural ooze. They suggested that small vessel bleeding in structures likely to be injured by a clip or cautery could readily be controlled by gelatin sponge. It was recommended as a seal for spinal fluid leaks, or to bridge a defect in the dura. A tendency for the absorbable sponge to adhere to dura and not to brain was noted. In another report, Light and Prentice[17] used absorbable sponge with thrombin as a "patch" for repairing venous sinuses. These authors reported that the lumen of the sinus was left patent without the formation of a thrombus within the vein. The method was inappropriate for arterial hemostasis.

Some contraindications to the use of absorbable sponge have appeared. Jenkins *et al.*,[18] for example, advised against its use in donor areas (skin graft) as liquefaction of the sponge interfered with after care.

METHODS AND TECHNIQUES.

Thrombin. Bovine thrombin is provided as a sterile water soluble white powder which remains stable in the dry state. In solution it retains its potency for about eight hours at room temperature. During this study, thrombin was supplied in vials containing 1000 units* and was diluted with sterile normal saline to various concentrations. In solution it was sprayed or used in the form of drops. In initial experiments as little as 100 u/cc were used, and in others as much as 1000 u/cc. The solution was impregnated in cotton pledget and wicks, gauze, or absorbable sponge.

In the process of coagulation, thrombin comes into contact with blood from a veno-capillary bed and initiates thrombosis by converting fibrinogen to fibrin. This produces a clot on the surface of the smaller vessels, but does not extend into the

*One unit is that amount which will cause one cubic centimeter of standardized fibrinogen solution to clot in fifteen seconds.

vessel.[19] As also suggested by Tidrick,[20] if much blood is present in the field, thrombin clots the surface blood and bleeding goes on under the clot. If the surface clot is wiped away, the "anchorage" is removed and bleeding may resume.

In this study, thrombin was painted over the medial surfaces of the septal flaps following submucous resection after the techniques described by Fox[21] and Reading,[22] was applied to the tonsillectomized fossae and to the nasopharynx after removal of adenoids and was packed into the mastoid or middle ear cavity following surgical exenteration of diseased bone. A cotton pledget soaked with thrombin was applied to the middle ear just prior to skin grafting.

Absorbable Gelatin Sponge. Absorbable gelatin sponge may be used in the dry state or saturated with saline, thrombin, or penicillin without prior neutralization. The dry sponge was trimmed to the desired size and thickness and shaped with a scissors using enough to cover the bleeding area or the defect. It was usually prepared for hemostasis by soaking in the desired solution and kneading forcefully until the air was completely expelled from the interstices. At this point the sponge was very limp and decreased in size. It was reinserted into the solution and allowed to swell to its original size and shape. Just prior to its application it is desirable to squeeze out the excess thrombin with a gauze sponge so that when it is applied over a bleeding point, blood is absorbed into its meshes where clotting occurs and the desired "anchorage" obtained. Where possible, it is advantageous to apply a cotton pledget over the absorbable sponge before applying suction in order to get blood into the interstices of the sponge. As emphasized by Light and Prentice[16,17] blood must flow into the sponge if good hemostasis is to be obtained.

When used in the ear, nose, and neck, absorbable gelatin sponge was saturated with thrombin containing 100-1000 u/cc. When the facial nerve was exposed or injured, during radical mastoidectomy, a longitudinal piece of absorbable sponge saturated in thrombin (500 u/cc) was placed over the nerve prior to skin grafting.

RESULTS.

THROMBIN.

A. Ear: During eighty radical mastoidectomies, thrombin (100-750 u/cc) was applied to bleeders from bone, soft tissue, and dura where epinephrine alone was tried and was not found to be effective consistently. Persistent bleeding over the posterior canal wall and facial nerve region was controlled frequently with thrombin. After radical mastoidectomy where primary skin grafts were done, cotton saturated with thrombin was packed into the middle ear and allowed to remain in place for ten minutes. This procedure has been found to leave a clean dry bed onto which a skin graft could be placed with excellent results. In some instances of active bone bleeding good hemostasis was not obtained with thrombin or epinephrine and a polishing burr was required.

B. Throat: Fifty tonsillectomies were performed by dissection and snare or Sluder techniques under ether anesthesia. All arterial bleeding and severe venous bleeding were controlled by ligatures. Thrombin (200 u/cc) on a cotton or gauze sponge was applied to one tonsillar fossa, and a dry sponge to the contra-lateral fossa. Satisfactory results were obtained in veno-capillary oozing with thrombin, but the dry sponge held in the control fossa also stopped veno-capillary oozing in the majority of the cases. Thrombin did not control heavy venous or arterial bleeding from the upper pole.

Post-operative bleeding occurred from a tonsillar fossa during the first 24 hours in one case where thrombin was applied to that fossa. In three instances delayed bleeding occurred without explanation from a tonsillar fossa on the 7-10th post-operative day.

C. Nasopharynx: Under ether anesthesia fifty adenoidectomies were performed using the La Force adenotome and curette. Thrombin on a gauze sponge (200-500 u/cc) was held against the visible bleeding adenoid bed. Active bleeding from the lateral wall of the nasopharynx was not controlled by thrombin. When capillary oozing occurred from the cut

nasopharyngeal epithelium or the adenoid bed, very prompt control of bleeding was obtained. In twenty-five routine cases, a one-inch post-nasal gauze pack saturated with thrombin (200 u/cc) was packed into the nasopharynx for 5-10 minutes immediately following adenoidectomy. The results were not superior to dry gauze packing.

D. Nose: In fifteen cases of epistaxis from Little's area and from the lateral nasal wall, thrombin solution (100-500 u/cc) was applied to the affected surface by spray or on a cotton pledget. In four cases prompt cessation of bleeding occurred; of these, three recurred in a short interval. Control of the bleeding was not obtained in the remaining cases.

Following six submucous resections, the medial surfaces of the septal flaps were painted and then the area flooded with thrombin (200 u/cc). The septal flaps were then firmly packed together with vaseline gauze packs for twenty-four hours. The application of thrombin in this surgical procedure may have contributed to a decrease in post-operative oozing and better adhesion of the flaps.

ABSORBABLE SPONGE WITH THROMBIN.

A. Ear.

Thirty radical endaural mastoidectomies were performed in which the Eustachian tube was cleaned out thoroughly. Absorbable sponge saturated in thrombin was packed into the common lumen of the Eustachian tube and canal of the tensor tympani muscle for the purpose of sealing off this lumen. This provided a good bed for a skin graft. In a few cases the absorbable sponge did not adhere satisfactorily in the Eustachian tube. As a result, the Eustachian tube lumen did not remain obliterated long enongh for the skin graft to "take."

In five radical mastoidectomies, unusually large cavities were produced in removing the diseased bone completely. It was considered desirable to reduce the size of the cavity in order to facilitate healing and post-operative care. These

recesses were packed with absorbable sponge containing thrombin and were skin grafted primarily or secondarily (five days). Excellent healing occurred. In another three cases, cells were removed posterior to the solid angle producing a large deep defect in the direction of the petrous apex. These tracts were filled with absorbable sponge and thrombin and skin grafted. Rapid healing took place.

Following the preparation of the membranous flap in the fenestration operation, a linear tear was obtained close to the tympanic membrane in four cases. After the flap was placed in position, a thin sheet of absorbable sponge was applied over the laceration and then covered with batiste gauze and the cavity packed in the usual manner. After the packing was taken out, the remaining sponge was easily removed by point suction, leaving the healed flap intact and in place.

In two cases where persistent and uncontrollable bleeding occurred from small vessels over the lateral sinus and dura, a small thin strip of absorbable sponge saturated with thrombin was applied to the bleeding point with control of the bleeding.

In nine cases where the facial nerve was found exposed by cholesteatoma, or where decompression was performed following operative injury to the nerve, a longitudinal piece of absorbable sponge containing thrombin was laid over the nerve to control bleeding and to act as a protective cushion for the skin graft to follow. There was no evidence in any of these cases which suggested interference with facial nerve function by absorbable gauze.

In one case where the dura in the area of the tegmen mastoidea was perforated with a burr during mastoidectomy, the dural perforation was sealed with absorbable sponge and thrombin. Excellent control of the spinal fluid ooze was obtained so that the operation could be completed and no immediate or late postoperative complications arose. In two cases where infected bone was removed from the hypotympanum over the jugular bulb, profuse bleeding was

encountered. Absorbable sponge saturated with thrombin was applied over the area and a pressure gauze packing placed on top of the absorbable sponge. Bleeding was controlled promptly. Gauze packing was removed on the fifth day without recurrence of bleeding and the area skin grafted uneventfully.

In one case of lateral sinus thrombosis,* following incision of the vessel wall and removal of the occluding thrombus, the proximal and distal portions of the vessel were packed off, and the site of incision was patched with absorbable sponge and thrombin and then packed with iodoform gauze. The packs were removed on the tenth day without recurrence of bleeding. In another case where needle aspiration of the exposed lateral sinus was performed in order to exclude an obliterating thrombus, brisk bleeding was readily controlled with absorbable sponge saturated with thrombin applied over the opening.

B. Nose.

Absorbable sponge with thrombin was used as an effective hemostatic adjunct in six out of seven cases where radical sinus surgery was performed.

1. A radical bilateral Killian operation was performed for osteomyelitis of the frontal bone. In exenterating the ethmoid sinuses a laceration of the dura was produced. Three pieces of absorbable sponge saturated with thrombin were placed over this opening. There was immediate cessation of spinal fluid flow and there were no post-operative complications while observed over a period of one year.

2. An exploratory external ethmoidectomy was performed for recurrent meningitis developing after a fracture through the ethmoidal labyrinth. A large dehiscence with surrounding soft bone was found extending through the ethmoid capsule into the anterior cranial fossa. During the removal of the involved bone a cerebrospinal leak occurred. Absorbable sponge saturated with thrombin was applied over the defect

*Case of Dr. A. M. Alden.

with immediate cessation of drainage. The patient has been observed for five weeks without complications.

3 and 4. Two external frontal operations (modified Killian)* were performed in cases of frontal empyema with secondary frontal lobe brain abscess. In both cases following the completion of the radical frontal sinus procedure, there was encountered a fistula into the frontal lobe from which pus exuded. After evacuation from the abscess cavity, absorbable sponge and thrombin were applied as a patch. The postoperative courses were entirely uneventful.

5. An external frontal operation (Killian)* was performed for osteomyelitis of the anterior table of the frontal sinus. After the completion of the sinus surgery, the superior and external angles of the sinus were obliterated with absorbable sponge and a gauze drain inserted into the medial angle of the wound. A fresh gauze drain saturated with penicillin was inserted daily until complete and satisfactory obliteration occurred.

6. A transantral ethmoidectomy was performed for an orbital decompression in a case of exophthalmos. A spinal fluid leak occurred in the region of the cribriform plate. Absorbable sponge and thrombin were placed up against the roof of the cavity. Spinal fluid continued to leak intermittently for ten days. Unsatisfactory sealing of this case was due in part to the inaccessibility of the lesion.

7. A radical Killian was done for empyema of the frontal sinus and a large defect found in the posterior plate. An area of granulation was present on the dura, in the center of which a small pinpoint defect was noted. The cerebrospinal leak was sealed with absorbable sponge containing 200 u. thrombin and 25,000 u. penicillin. The dead space produced by the extensive removal of bone was obliterated with absorbable sponge and thrombin. The results here appeared good six months after operation.

*Cases of Dr. M. F. Arbuckle.

In one case where contiguous perforations of both mucous membrane flaps were obtained following submucous resection of the nasal septum, a thin dry flat sheet of absorbable sponge was inserted between the membranes so as to obliterate the defect and prevent "turning in" of the edges. Excellent healing followed and no unusual residual thickening resulted. After one month no perforation had appeared. In a similar case* where a cartilage implant was to be used, it was possible to suture one side, insert the cartilage and then over this the sheet of dry sponge. The cartilage was not extruded, and good healing occurred.

In two cases where biopsies were taken from Little's area (varicosity and neuroma), large excisions of mucous membrane were required. Persistent oozing was controlled by absorbable sponge and thrombin with an overlying pressure gauze pack. After several days the absorbable sponge was easily blown out of the nose without recurrence of bleeding. The sponge acted as a protective covering so that removal of the gauze packing did not cause further bleeding.

In ten cases of epistaxis, absorbable sponge and thrombin were used. Bleeding occurred from the inferior, middle, and superior meatuses and from Little's area. It was found difficult to apply the sponge in those cases where bleeding arose from the lateral nasal wall, particularly when the bleeding site was posterior. Good results were obtained in one case involving Little's area and the two instances from the inferior meatus.

C. Throat and Nasopharynx.

It was not considered advisable to use absorbable sponge following tonsillectomy and adenoidectomy since no method could be devised which insured against the danger of aspiration of the sponge.

D. Neck.

Absorbable sponge and thrombin were used during four radical dissections where bleeding could not be satisfactorily

*Case of Dr. Guerdan Hardy.

controlled with ligatures. These cases received pre-operative X-ray therapy which undoubtedly contributed to the difficulty of hemostasis. Prompt control of bleeding was obtained in all cases and no satisfactory post-operative sequelae occurred.

DISCUSSION.

In this study no attempt was made to compare the several biological hemostatics now available. The relative merits and applicability of these agents will form the basis of a future report.

The mechanism of the action of thrombin has been widely discussed.[19] It is said to exert its effect by clotting the surface blood which extends over the openings of the vessels forming an "anchorage clot" and must not be disturbed or bleeding will recur. This is in contrast to epinephrine which exerts its effect by vasoconstriction. When the constrictive action of the epinephrine wears off, there is slow vasodilation. If clotting is not adequate, bleeding will recur. In other words, epinephrine produces its effect by constricting the vessel and thrombin by forming a clot over the opening in the vessel wall.

Epinephrine is desirable for effective temporary hemostasis while cleaning out thickened diseased membrane in the middle ear. A cotton tipped applicator soaked in epinephrine is of value in removing granulations from the horizontal semicircular canal and for sweeping infected mucous membrane into the middle ear in order to identify the facial nerve. It is clear that the formation of a surface blood clot by the use of thrombin would not be of value in this instance.

After cleaning out the middle ear and Eustachian tube, a completely dry field is desirable for skin grafting. The application of thrombin in this case leaves a very satisfactory dry clotted bed. Thrombin may also be of particular value for dural oozing, bleeding over the facial nerve canal and where cautery or polishing burr may be inadvisable.

It appears that thrombin is effective for the control of persistent veno-capillary oozing. In the tonsillar fossa and adenoid bed following tonsillectomy and adenoidectomy, thrombin may be valuable where there is persistent veno-capillary ooze from muscle or mucous membrane. This is of particular importance where further manipulation and efforts to ligate the bleeding points appear to increase the bleeding.

From both a theoretical and a practical point of view, the use of thrombin spray in the nose appears ineffective. Within our limited experience, we agree that thrombin may be of value for its adhesive and hemostatic qualities when applied to the septal flaps following submucous resections, particularly if it is desirable to avoid the use of nasal packing.

Our experience in this study does not permit definitive conclusions as to the optimum concentration of thrombin for adequate hemostasis. It has been our practice, in most circumstances, to use 500 u/cc, and no contraindications to this concentration have been found.

Absorbable sponge saturated with thrombin was observed to have special application in otologic and sinus surgery. The ease with which it can be prepared, molded, and applied to the ear makes it superior for use in ear surgery. During radical mastoidectomy it is necessary to occlude the Eustachian tube in order to prevent Eustachian discharge from entering the radical cavity and also to provide a smooth bed for skin grafting. Unfortunately, the physical characteristic of absorbable sponge makes it difficult to manipulate in the restricted openings and some failure to adhere to the canal walls is reported. On the other hand, absorbable sponge was readily packed into the open recesses of the mastoid cavity following exenteration of cell tracts or where there was an unusually large mastoid cavity created by the necessity of removing a great deal of infected bone. In these cases it appeared desirable to reduce the size and round out the cavity over which the skin graft was to be applied. It is believed that more rapid healing occurred and post-operative care was simplified.

Where a radical mastoidectomy was performed, and the facial nerve was found exposed by disease or was decompressed following injury, absorbable sponge was used as a protective cushion over the facial nerve. It is believed that this procedure served to prevent direct apposition of skin graft to the nerve, and avoided undue pressure on the nerve.

Using the patch technique absorbable sponge saturated with thrombin was applied with apparent impunity over lacerations or fistulas into the dura. In those surgical cases where accidental laceration occurred and there was a spontaneous flow of spinal fluid, there was effective control with a single exception. In this instance an attempt was made to control spinal fluid leakage from the roof of the sinus cavity without success.

Where there was a flow of purulent secretion through a dural fistula (frontal lobe abscess), an absorbable sponge was applied. It would appear that the sealing off of a fistula into an infected intracranial lesion would be contrary to good surgical principles, yet the post-operative results in two cases reported have been strikingly good. In these cases it was impossible to ascertain whether the patch remained intact since there was no opportunity to re-examine the lesion post-operatively.

It appears from the literature that absorbable sponge may allow for healing of a vessel wall without formation of an occluding mural thrombus. If absorbable sponge is applied over the incision in the lateral sinus after the bleeding is controlled by packing both ends of the exposed lateral sinus, it is conceivable that upon removal of the packs secondary hemorrhage may be prevented, and may allow for more rapid recanalization. Further data are necessary on this point.

If a vigorous bleeding point in the nose is controlled by packing with vaseline gauze and the gauze removed at a later date, bleeding may recur as a result of disturbing the blood clot. It appears reasonable to assume that if the bleeding point is first covered with an absorbable sponge and then

pressure with surgical gauze is superimposed, the bleeding point is not only controlled by the pressure, but when the pack is removed later, the "anchorage clot" and protective sponge covering remains undisturbed and the bleeding may not recur. In the nose, where it was possible to visualize the bleeding point, such a procedure was followed with some good results. In the majority of cases, particularly where the bleeding point was posterior and lateral in the nose, some difficulty was encountered. The physical characteristics of the absorbable sponge and limited space within the nose made it difficult to apply the sponge and to obtain sufficient counter-pressure.

Although the good report on the use of absorbable sponge as an "intraseptal bridge" in cases of through and through perforation following submucous resection is limited to two cases, it is believed worthy of further observation and investigation as a simple method of avoiding a troublesome and sometimes difficult post-operative problem.

CONCLUSIONS.

1. Thrombin was useful in controlling veno-capillary oozing after tonsillectomy and adenoidectomy. Vigorous venous and arterial bleeding were not controlled by the use of thrombin.

2. The application of thrombin may be of value in the control of veno-capillary ooze from the nose, but brisk bleeding from Little's area or from the lateral nasal wall was not controlled.

3. Thrombin, when applied to the middle ear following radical mastoidectomy, provided a dry bed for skin grafting. It was effective in cases of bone and soft tissue oozing, dural and lateral sinus bleeding where epinephrine failed and where cautery was deemed inadvisable.

4. Absorbable sponge saturated with thrombin was demonstrated to be an innocuous and valuable adjunct for use in the following surgical procedures:

a. Good results were obtained during radical mastoidectomy, *e.g.*, obliteration of inaccessible parts of the mastoid cavity prior to skin grafting.

b. It was found useful to patch lacerations and incisions of the dura, jugular bulb, and lateral sinus.

c. It was used successfully as an innocuous hemostatic agent as well as a protective cushion over the exposed facial nerve.

d. The sponge was used to protect linear tears in the membranous flap during the fenestration operation.

e. Good results were obtained in radical neck dissection where unusual bleeding followed X-ray therapy.

f. Promising results were obtained in the use of a thin sheet of sponge as an "intraseptal bridge" in cases of post-operative through and through perforations of the nasal septum.

g. Unsatisfactory results were obtained when the sponge was used in an attempt to control brisk bleeding from the lateral nasal wall.

5. No methods could be devised for the safe use of absorbable sponge in the oropharynx or nasopharynx.

BIBLIOGRAPHY.

1. Ogura, J. H., and Senturia, B. H.: Epistaxis. The Laryngoscope, 59:7:743-763, July, 1949.

2. Cushing, Harvey: The Control of Bleeding in Operations for Brain Tumors. With the Description of Silver "Clips" for the Occlusion of Vessels Inaccessible to the Ligature. *Ann. Surg.*, 54:1-19, July, 1911.

3. Mellanby, J.: Thrombase: Its Preparation and Properties. *Proc. Roy. Soc.*, London, Series B, 113:93-106, June 11, 1933.

4. Seegers, W. H.; Brinkhous, K. M.; Smith, H. P., and Warner, E. D.: The Purification of Thrombin. *Jour. Biol. Chem.*, 126:91-95, Nov., 1938.

5. Bering, E. A., Jr.: Chemical, Clinical and Immunologic Studies on the Products of Human Plasma Fractionation. XX.—The Development of Fibrin Foam as a Hemostatic Agent and for Use in Conjunction with Human Thrombin. *Jour. Clin. Inves.*, 23:586-590, July, 1944.

6. CORRELL, J. T.; PRENTICE, H. R., and WISE, E. C.: Biologic Investigations of a New Absorbable Sponge. *Surg. Gynec. and Obst.*, 81:585-589, 1945.

7. FRANTZ, V. K.: Absorbable Cotton, Paper and Gauze (Oxidized Cellulose). *Ann. Surg.*, 118:116-126, July, 1943.

8. SPITZ, E. B.; ZIFF, M.; BRENNER, C., and DAVISON, C.: A New Absorbable Material for Use in Neurological and General Surgery. *Sci.*, 102:621-622, Dec. 14, 1945.

9. BICE, C. W.; MACMASTERS, M. M., and HILBERT, G. E.: Proposed Use of Starch Sponges as Internal Surgical Dressings Absorbable by the Body. *Sci.*, 100:227-228, Sept. 8, 1944.

10. SEEGERS, W. H., and MCGINTY, D. A.: Further Purification of Thrombin: Probable Purity of Products. *Jour. Biol. Chem.*, 146:511-518, Dec., 1942.

11. SEEGERS, W. H.; WARNER, E. D., BRINKHOUS, K. M., and SMITH, H. P.: The Use of Purified Thrombin as an Hemostatic Agent. *Sci.*, 89:86, Jan. 27, 1939.

12. TIDRICK, R. T.; SEEGERS, W. H., and WARNER, E. D.: Clinical Experience with Thrombin as an Hemostatic Agent. *Surg.*, 14:191-196, Aug., 1943.

13. CORRELL, J. T. and WISE, E. C.: Certain Properties of a New Physiologically Absorbable Sponge. *Proc. Soc. Exper. Biol. and Med.*, 58:233-235, 1945.

14. CORRELL, J. T., and WISE, E. C.: The Stability of Thrombin, Penicillin, or Streptomycin, in the Presence of Gelatin Sponge, Oxidized Cellulose or Each Other. *Surg., Gynec. and Obst.*, 85:211-213, Aug., 1947.

15. JENKINS, H. P., and CLARKE, J. S.: Gelatin Sponge, a New Hemostatic Substance. Studies on Absorbability. *Arch. Surg.*, 51:253-261, Nov.-Dec., 1945.

16. PILCHER, C., and MEACHAM, W. F.: Absorbable Gelatin Sponge and Thrombin for Hemostasis in Neurosurgery. *Surg., Gynec. and Obst.*, 81: 365-369, 1945.

17. LIGHT, R. U., and PRENTICE, H. R.: Gelatin Sponge: Surgical Investigation of a New Matrix Used in Conjunction with Thrombin in Hemostasis. *Arch. Surg.*, 51:69-77, Sept., 1945.

18. LIGHT, R. U., and PRENTICE, H. R.: Surgical Investigation of a New Absorbable Sponge Derived from Gelatin for Use in Hemostasis. *Jour. Neurosurg.*, 2:435-455, Sept., 1945.

19. JENKINS, H. P.; SENZ, E. H.; OWEN, H. W., and JAMPOLIS, R. W.: Present Status of Gelatin Sponge for the Control of Hemorrhage. *Jour. A. M. A.*, 132:614-619, Nov. 16, 1946.

20. SEEGERS, W. H., and SHAY, E. A.: Hemostatic Agents. Chas. C. Thomas, 1948. Chap. 2 (Mechanics of Topical Hemostasis with Thrombin).

21. TIDRICK, R. T.: Recent Advances in Surgical Hemostasis: Evaluation of Absorbable Agents. *Tran. Amer. Acad. Ophthal. and Otol.*, Sept.-Oct., 1947.

22. FOX, S. L.: Use of Thrombin in Rhinologic Surgery. THE LARYNGOSCOPE, 56:48, 1946.

23. READING, P.: Some Uses of Thrombin in the Surgery of the Ear, Nose, and Throat. *Brit. Med. Jour.*, 2:295-296, 1946.

V. LIX
10/49
#10

CLINICAL RELIABILITY OF BONE CONDUCTION AUDIOMETRY.*

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– I –

Bone conduction audiometry has received much criticism in past years. Otologists and audiologists distrust thresholds obtained by bone conduction because of fear that the methodology possesses two limitations. The first supposed limitation is lack of *validity*, or inadequacy of the norms for bone conduction because of variations introduced by differences in skull structure, positioning of the vibrator, and other factors. The second supposed limitation is doubtful *reliability*, or excessive discrepancy between test and retest measurements due to positioning, vibrator pressure, and related influences. The value of bone conduction audiometry as a diagnostic aid must remain in doubt until research has clarified the validity and reliability which may be expected when bone conduction thresholds are obtained under clinical conditions.

Several investigators have dealt with aspects of bone conduction audiometry which are pertinent to the present study. These contributors include Witting and Hughson,[8] Lierle and Reger,[5] McNally and his associates,[6] Senturia and Thea,[7] Greenbaum, Kerridge and Ross,[3] and Beasley in the National Health Survey.[1] The work of these men leads to two beliefs: *1.* that bone conduction measurements on normal ears show only slightly greater variability from the theoretical normal

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curve than is shown by the comparable air conduction thresholds; *2.* that bone conduction thresholds are reasonably reliable; however, there appears to be little evidence bearing on the question of how closely a patient's bone conduction thresholds *on repeated tests* may be expected to agree with one another.

Reasonable interpretation of an audiogram obtained under clinical conditions cannot be made until information is available on this latter point. The margin of uncertainty for bone conduction audiometry, as contrasted to the margin of uncertainty for air conduction thresholds, must first be defined. It is this topic with which the present investigation deals. Specifically, the primary aim was to compare test-retest reliability on air and bone conduction results as gathered in a clinical situation on a heterogeneous population of patients with hearing loss. Thus, the study is concerned purely with the question of *reproducibility of thresholds.* The study is not concerned with the question of validity. In other words, it is not concerned with whether observed thresholds are true representations of the amount of hearing loss. This latter topic is obviously important, but it is beyond the scope of the investigation here reported.

– II –

The data forming the basis for this investigation were audiometric records taken from the files of the Aural Rehabilitation Program at Deshon General Hospital. Two series of audiograms were compared: namely, an initial audiogram and a second audiogram for each patient in the group. Within the limits of the Deshon population, the data represented an unselected sampling in terms of age, duration of hearing loss, audiometric pattern, type of hearing loss, linguistic ability, and intelligence; however, Army regulations were such that very few patients with near-normal hearing were included. Otherwise, the degree of loss also varied widely.

The audiometric data were drawn alphabetically from the Deshon files. but only records meeting the following criteria were selected.

1. The initial test and the retest must have been administered with the same audiometer. (This precaution eliminated variations in threshold due to differences between instruments.)

2. The two tests must have been given approximately a month apart. (This precaution reduced differences which might be due to progressive deterioration in the hearing of some patients.)

3. Measurable thresholds must have been obtained on at least three frequencies by both air and bone modalities, as well as on both tests. (This requirement was necessary to yield a reasonable number of test-retest comparisons for each frequency investigated.)

4. All tests must have been administered between November, 1944, and May, 1946. (This time limitation was imposed because improved standardization of bone conduction testing was available at Deshon between these dates.)

5. The same masking technique must have been employed on both tests. (This precaution guarded against large differences, particularly in bone conduction thresholds, which might be due to variations in masking.)

6. Cases were ruled out if their records suggested either a psychogenic or a central factor. (This precaution guarded against the large test-retest variations which are typical when such factors are present, and which may not be considered as indicative of the reliability of the audiometric technique *per se*.

The study was limited by original plan to evaluate data on 250 patients (500 ears). It developed that the criteria were sufficiently rigid, particularly in the requirement that the same audiometer be used for both tests, so that almost the entire case file had to be surveyed to extract the 250 sets of data.

– III –

The group under investigation encompassed wide ranges in both degree and contour of hearing loss. This fact is illustrated in part by Fig. 1, which summarized the monaural averages (512-2048 c.p.s.) revealed by the initial audiograms for the 500 ears under investigation. The air conduction averages evidenced the wider spread by ranging from —5 db to 90 db of hearing loss, with a mean loss of 43 db and a standard deviation of 14.7 db. The bone conduction average ranged from —10 db to 55 db, with a mean loss of 15 db and a standard deviation of 13.6 db; however, as can be seen from Fig. 1, the distributions for both sets of scores were somewhat skewed, the air conduction scores being skewed negatively and the bone conduction positively.

The patients were predominantly young males, but their ages ranged from 19 to 62 years.

The distribution of the diagnoses supplied by the medical officers were:

Conductive loss	79
Mixed loss—predominantly conductive	37
Mixed loss	61
Mixed loss—predominantly perceptive	14
Perceptive loss	55
Normal hearing	4
Total	250 cases

The group was limited to men with reasonable understanding for speech. It seems safe to assume that otherwise the range of intelligence and linguistic ability of the group was great, although no quantitative data on these factors were at hand.

– IV –

The audiometric technique employed at Deshon was the ascending method advocated by Hughson and Westlake.[4] Careful rather than rapid testing was emphasized. One half-hour was allotted for each pure tone test.

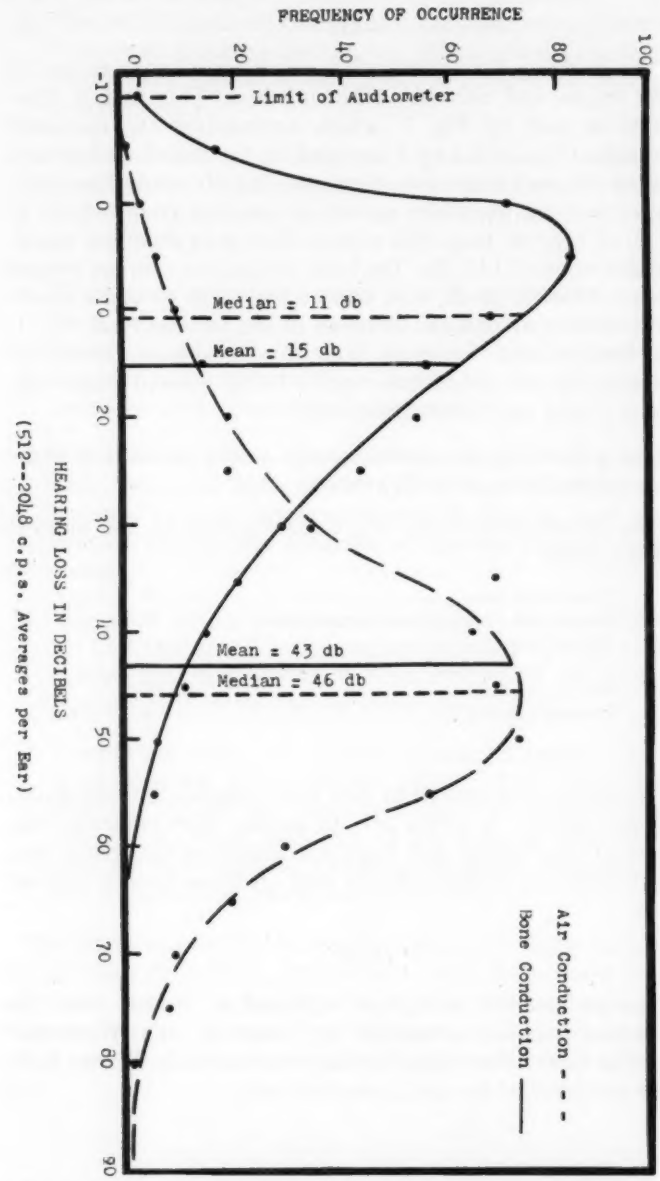

Fig. 1. Degree of hearing loss by air conduction and bone conduction for monaural averages from 512 c.p.s. to 2048 c.p.s. Determined from initial audiograms for 500 ears.

The tests were administered in special chambers with superior acoustic characteristics.

Three types of commercial audiometers were employed routinely: namely, the Western Electric 6B, the Sonotone 20, and the Maico D5. The Western Electric and Sonotone instruments were used as supplied by the manufacturers; *i.e.*, each with its original air conduction and bone conduction receivers. The same was true for the air conduction testing performed with the Maico instruments. Here, however, bone thresholds were obtained with an auxiliary unit activated by the audiometer. This auxiliary system consisted of an amplifier, a matching transformer, a meter, and a bone conduction receiver of the hearing aid type held in a steel headband.

The auxiliary bone unit was calibrated by testing normal persons in a special sound-proof room. Once the calibration was determined, it was possible to standardize the entire system relative to the attenuator scale on the audiometer. This was done by adjusting the output of the amplifier to yield the required voltage as read on the meter. Obviously this voltage varied from one frequency to another — since the characteristics of the circuits, the bone oscillator and the human mechanism were involved; however, once the standard had been determined, conditions could be reproduced at will. From this point onward, audiometry proceeded according to the ascending threshold technique.

With the exception of the auxiliary system just described, thresholds (for both air and bone) were read as calibrated by the respective manufacturer; however, these calibrations were routinely checked against normal ears and any discrepancies corrected.

– V –

The data under investigation incorporate uncontrolled variables of several types. Factors such as age, pattern of hearing loss, and duration of loss have already been mentioned. Another variable involved actual fluctuations in some patients' hearing between the initial test and the retest. Then, too, five

audiometrists administered the tests under consideration. All five were well trained and used the same testing technique; however, no attempt was made to select only cases where the same audiometrist administered the two tests for a particular patient. It was felt that a somewhat more realistic estimate of the clinical reliability would be forthcoming if the retest were administered by a person not having prior knowledge of the patient's hearing.

Stated differently, the criteria for selection of cases were chosen with the view of minimizing those variables in the Deshon situation which might obscure the basic issue: namely, the test-retest reliability of bone conduction thresholds. By contrast, variables which were deemed inherent in clinical practice were ignored in the criteria for selection. The assumption here was that, although failure to control such variables would reduce reliability in comparison to that of the laboratory situation, the results would constitute a fairer indication of the clinical efficiency of bone conduction audiometry. The simultaneous exploration of reliability for air conduction thresholds furnished a further reference against which to evaluate the findings for bone conduction.

– VI –

The statistical treatment of the data involved analysis of a series of Difference Scores derived from the audiometric data. The procedure in computing each Difference Score was to subtract the appropriate threshold reading on the retest from the corresponding reading on the initial test. Thus, each Difference Score was a direct numerical statement of the magnitude and direction of discrepancy between the test-retest readings. Specifically, *1.* a positive Difference Score represented a poorer threshold on the first test than on the second one; *2.* a negative Difference Score indicated the reverse; and *3.* a Difference Score of zero was obtained when the companion thresholds were identical on both tests. The procedure eliminated from consideration the amount of loss rep-

resented by a particular threshold and centered upon test-retest agreement or discrepancy.

Difference Scores were computed for each test frequency from 256 through 4096 c.p.s.* In addition, average (or composite) Difference Scores for the 256-4096 c.p.s. range were obtained; furthermore, these various sets of Difference Scores were computed from both the air conduction and bone conduction data.

The various sets of Difference Scores were compared with one another statistically. To this end, the mean Difference Score and the standard deviation of Scores were determined for each set. Next, **t** tests were employed to estimate the statistical significance of divergencies in the means and standard deviations for the various sets of scores;[2] moreover, the correlation between air and bone results (for individual frequencies and for the composite of all frequencies) was computed by the product-moment method.

The results of the statistical analyses are discussed below under five headings: namely, *1.* central tendencies of the data, *2.* variability of the test-retest comparisons, *3.* correlation between threshold shifts for air and bone conduction, *4.* intra-frequency variations in test-retest reliability, and *5.* variation in test-retest reliability with different audiometers.

– VII –

As can be seen from Table I, all of the means of Difference Scores for air conduction were close to zero both for each frequency considered separately and in the composite. The greatest negative diverge was yielded by the data for 512 c.p.s. (—.55 db), while 4096 c.p.s. yielded the largest positive mean (.35 db). Other values ranged between these extremes. Similar findings emerged for bone conduction. Here the range of means was from —.25 db (for 1024 c.p.s.) to .55 db (for 2048 c.p.s.). Thus, the central tendencies for both air and

*Only frequencies from 256 through 4096 c.p.s. were considered because the available data on bone conduction thresholds were limited to this range.

bone conduction fall within a very limited range. These findings favor the assumption that uncontrolled variables were randomly distributed. For a given set of thresholds, the shifts in one direction (from test to retest) were almost completely counterbalanced by shifts in the other direction.

TABLE I.

Mean Difference Scores, in DB, Derived from Test-Retest Thresholds for Air Conduction and, Independently, for Bone Conduction. Also t Scores Indicating Significance of Discrepancies in Means for Air and Bone.

Frequency	256	512	1024	2048	4096	Composite
No. of Comparisons	495	498	499	482	403	2377
Air	—.15	—.55	—.30	.25	.35	—.10
Bone	.40	.15	—.25	.55	.10	.20
t Scores*	1.265	1.528	.208	.611	.429	1.311

*t Score of 1.966 and 2.588 significant at 5% and 1% level.

Table I also summarized t scores derived when the means for air conduction were compared with those for bone conduction. The largest t score was 1.528 for 512 c.p.s. (where 1.966 is required for 5 per cent significance). Hence, neither the results for individual frequencies nor for the composite may be interpreted as indicating a significant difference between the central tendencies for air conduction and bone conduction. In other words, there was no evidence of a systematic variation, due to the modality of stimulation, in the test-retest counterbalance of variables in the Deshon situation.

The foregoing findings confirm the belief that the data under consideration are adequate for purposes of the present study. No statistical evidence emerged which suggested systematic differences between first and second tests. Neither was there any statistical evidence of significant dissimilarity in the central tendencies for the air and the bone conduction modalities. Finally, the results lead to the conclusion that, for the group as a whole, the general level of hearing loss did not change between the first and second tests.

– VIII –

The standard deviations of the several sets of Difference Scores were used as the basis for estimating test-retest variability. The comparative sizes of these standard deviations give an indication of the relative reliabilities of thresholds at different frequencies and for the two different test modalities.

The standard deviations computed from the present data are summarized in Table II. One notes that, for the air conduction data, the standard deviations for individual frequencies ranged from 8.35 db to 9.70 db. The smallest standard deviations were for frequencies 512 to 2048 c.p.s., with the least variability falling at 1024 c.p.s. This finding is not an unexpected one in the light of earlier studies, and it will be discussed later. The standard deviation, by air conduction, for the composite of all frequencies was 9.10 db.

The variability recorded in Table II is considerably greater than the 5 db reported by Witting and Hughson,[8] who made a total of 297 tests on 17 patients under strict research conditions. One concludes that the reliability of the audiometry conducted at Deshon was less than achievable under more exacting control of test conditions; however, the reliability of the methodology itself was probably somewhat better than the figures in Table II would seem to indicate because such factors as actual changes, between tests, in threshold acuity contributed to the sizes of the standard deviations.

TABLE II.

Standard Deviation of Difference Scores, in DB, Derived from Test-Retest Thresholds for Air Conduction and, Independently, for Bone Conduction. Also t Scores Indicating Significance of Discrepancies in Standard Deviations for Air and Bone. Plus Coefficient of Correlation Between Difference Scores for Air and Bone.

Frequency	256	512	1024	2048	4096	Composite
No. of Comparisons	495	498	499	482	403	2377
S. D. Air	9.25	9.15	8.35	9.15	9.70	9.10
S. D. Bone	7.70	8.35	7.65	9.05	9.40	8.45
t Scores*	4.394	2.282	2.159	.192	.600	4.061
Coeff. Corr.	.35	.33	.36	.33	.32	.34

*t Score of 1.966 and 2.588 significant at 5% and 1% levels.

Turning to the results for bone conduction, one notes that the standard deviations ranged from 7.65 db to 9.40 db for the various test frequencies, with 8.45 db as the composite. Again the smallest value was that for 1024 c.p.s., and with this exception the variability tended to increase as the test frequency became higher.*

Table II also reveals that the standard deviations of the Difference Scores by air conduction were all larger than the standard deviation for the same frequency by bone conduction. In other words, the present data did not reveal as much variability in retesting bone conduction thresholds as in retesting the corresponding air conduction thresholds. This finding is in conflict with the general belief in the field and is contrary to the expectation which the authors held prior to undertaking the study.

Fuller interpretation of the finding just mentioned was possible through the application of **t** tests, which allowed one to estimate the significance of observed differences in the standard deviations for air and bone. Table II also presents the results of these **t** tests. The **t** scores for 2048 and 4096 c.p.s. (0.192 and 0.600, respectively) fall substantially below the level of statistical significance. One concludes that, for these two frequencies, the reliability of air conduction and bone conduction testing was approximately equivalent in the population here studied. By contrast, the **t** scores for 256, 512, and 1024 c.p.s. (4.394, 2.282 and 2.159, respectively) reached statistical significance.† In each of these instances the standard deviation for air conduction was larger than that for bone conduction. Similarly, the standard deviation was smaller by bone for the composite of all frequencies. Here the **t** score (4.061) exceeded 1 per cent significance.

*The small standard deviation for bone at 256 c.p.s. is provably in part an artifact. Many cases, at this frequency, obtained normal or better than normal thresholds on the first test. These cases were limited in the measurable retest variation which could be observed when the shift was in the direction of hyperacuity. Had this restriction not been operative, the variability for 256 c.p.s. would probably have been found to be greater than here reported.

†Actually, although the **t** score for 256 c.p.s. most exceeded the 1 per cent level of significance, the probable artifact already mentioned undoubtedly accounted in part for the magnitude of the **t** score.

One might conclude that the above findings reveal bone conduction measurement to have been more reliable than air conduction measurements; however, this conclusion needs modification because the total intensity range of thresholds by bone conduction was 70 db. (from 10 db better than normal to a loss of 60 db) while the range for air was 110 db (from 10 db better than normal to a loss of 100 db).* Since the criteria for case selection required that most thresholds fall within these ranges on both tests, the end-result would be a comparative restriction in the number of extreme variations to be found in the data for bone conduction. The statistical influence would be a decrease in the standard deviation; therefore, the most reasonable interpretation of the results reported above seems to be that in the Deshon situation, the test-retest reliability of bone conduction thresholds was equivalent to the reliability for air conduction. One concludes: *the common opinion that bone conduction thresholds are substantially less repeatable than air conduction thresholds does not apply when reasonable precautions are taken to insure control of the bone conduction technique.*

– IX –

The degree to which the data for air and bone conduction evidenced parallelism, in the threshold difference between initial test and retest, was estimated by the product-moment method. Table II summarizes the coefficients of correlation which were obtained. All correlations were of the order of $r = +.33$. Coefficients of this magnitude may be interpreted as showing a low but positive parallelism. In other words, individual patients evidenced a tendency to yield Difference Scores for air conduction and for bone conduction which were of like sign and of like magnitude, although many subjects deviated from this pattern.

Assuming this finding to be evidence that true fluctuations in hearing were involved in the data, the variabilities *due to*

*The reader will recognize that these ranges varied somewhat with test frequency and with the make of audiometer, but this fact is of secondary importance at the moment.

the audiometric technique itself are probably more accurately represented by the standard errors of estimate.* Table III reports the standard errors of estimate for the different test frequencies and the two test modalities. One notes that these standard errors range from 7.15 to 9.18 db. Thus, test-retest variability remained relatively large in the Deshon situation even after a reasonable correction for probable fluctuations in patients' acuity.

TABLE III.

Standard Error of the Estimate of Difference Score, in DB.

Frequency 256	512	1024	2048	4096	Composite
Air Conduction.....8.70	8.65	7.82	8.65	9.18	8.58
Bone Conduction..7.24	7.90	7.15	8.55	8.90	7.94

– X –

The comparative variability of thresholds, from one frequency to another, was estimated by t tests applied to the standard deviations of the Difference Scores. Tables IV and V report the results for air conduction and bone conduction, respectively.

The only significant t scores in Table IV are those in which 1024 c.p.s. was compared with the other test frequencies. Thus, 1024 c.p.s. is the only frequency which was found to be significantly less variable than any other frequency when tested by air conduction. This finding agrees with the conclusion of Witting and Hughson that the inherent error in repeating audiograms is a function of frequency and that 1024 c.p.s. yields "maximum ease of listening."[8]

A shown by Table V, similar results emerged for bone conduction. Here, however, 256 c.p.s. was not distinguished significantly from 1024 c.p.s.,† while 4096 c.p.s. was significantly separated from all other frequencies but 2048 c.p.s. Thus, the

*These estimates are derived by revising the standard deviations in terms of the coefficients of correlation.[2]

†This finding was probably due to a spurious reduction in variability for 256 c.p.s., which has been already mentioned as a likely artifact inherent in the data.

dependence of the inherent error upon frequency appears, on the basis of the data at hand, to be more pronounced for bone conduction thresholds than for air. Most noteworthy is the fact that, in general, thresholds for 1024 c.p.s. again seemed to be more precise than those for other frequencies.

TABLE IV.

t Scores Derived from Comparison of the Standard Deviations of Air Conduction Difference Scores for the Various Test Frequencies.

Frequency	512	1024	2048	4096
256	.238	2.308	.238	1.011
512		2.051	.000	1.236
1028			2.051	3.034
2048				1.236

t Scores of 1.966 and 2.588 are significant at 5% and 1% level. Number of Difference Scores ranged from 499 to 403 (see Table I).

TABLE V.

t Scores Derived from Comparison of the Standard Deviations of Bone Conduction Difference Scores for the Various Test Frequencies.

Frequency	512	1024	2048	4096
256	1.831	.1408	3.462	4.048
512		1.972	1.795	2.500
1024			3.590	4.167
2048				.787

t Scores of 1.966 and 2.588 are significant at 5% and 1% level. Number of Difference Scores ranged from 499 to 403 (see Table I).

– XI –

A comparison was made of the standard deviations of Difference Scores obtained with two of the seven audiometers involved in the present investigation. The purpose was to determine whether the audiometer influenced the reliability of test-retest scores.

The two audiometers being compared were of different makes. They are here designated as instrument X and instrument Y, since the writers do not feel justified in drawing

conclusions regarding the relative efficiencies of types of instrument from a single representative of each kind.

TABLE VI.

Standard Deviations for Composite of All Frequencies, for the Air Conduction and Bone Conduction Difference Scores Obtained with Two Different Audiometers. Also t Scores Indicating Significance of Discrepancies in Standard Deviations for the Two Audiometers. Standard Deviations Reported in DB.

	Audiometer X 108 Ears	Audiometer Y 154 Ears	t Score*
S. D. Air	10.95	8.10	4.64
S. D. Bone	9.85	6.95	3.75

*A t score of 3.25 is here significant at 1% level.

Findings are reported in Table VI. Instrument X yielded larger standard deviations, for both air conduction and bone conduction measurements, than did Instrument Y. For both modalities of stimulation the t scores exceeded the 1 per cent level of statistical significance. One concludes that the reliability of pure tone thresholds is partially dependent upon the particular audiometer used; however, this conclusion does not contradict the earlier finding that, under proper control, bone conduction thresholds have as great test-retest reliability as air conduction thresholds.

– XII –

In summary: the present study was a statistical investigation of the comparative reproducibility, *i.e.*, test-retest reliability, of air conduction and bone conduction thresholds. The study was not concerned with the question of validity, *i.e.*, the adequacy of the norms for the two modalities of stimulation; furthermore, the study was based upon tests administered under clinical conditions.

Sets of data on 250 cases were selected from the records of the Aural Rehabilitation Program at Deshon General Hospital. Each set consisted of an initial audiogram and a second audiogram obtained approximately a month later. Selection

was limited to test results in which both the air conduction and bone conduction measurements were reasonably complete. Other restrictions, such as the requirement that both tests must have been given with the same audiometer, were observed in the selection of data.

The 250 cases represented the major types of auditory impairment and encompassed a wide range in degree of loss. The average loss (over the 512-2048 c.p.s. range) was 43 db by air conduction and 15 db by bone conduction.

Seven commercial audiometers, some with special adaptations to improve bone conduction testing, were used in administering the tests under consideration. All bone oscillators were of the hearing aid variety.

Test-retest agreement, both for individual frequencies and in the aggregate, was expressed in Difference Scores which represented the degrees and directions of discrepancies between the two tests. The Difference Scores were subjected to statistical comparisons. The comparisons included determination of the means and standard deviations of Difference Scores, estimation of the significances of divergencies through **t** tests, and computation of correlations between the Difference Scores for air conduction and bone conduction. All comparisons were based upon the number of ears for which appropriate Difference Scores were available.

The main findings and conclusions were as follows:

1. The means of all Difference Scores approximated zero and the corresponding **t** tests were non-significant. Thus, for a given set of thresholds, the shifts in one direction were almost completely counterbalanced by shifts in the opposite direction. For the group as a whole, the general level of hearing did not change between the first and second tests, and the data were adequate for the purposes at hand.

2. The standard deviations for the air conduction data were somewhat larger than for the corresponding bone

conduction measurements. The discrepancy reached statistical significance for 256, 512, and 1024 c.p.s., as well as for the composite of all test frequencies. In other words, the test-retest reliability of bone conduction thresholds was here found to equal or exceed that for air conduction measurements. It follows that *the repeat accuracy of bone conduction measurements may be as good as that for air conduction thresholds when appropriate precautions are taken in testing.*

3. Low but positive parallelism between air and bone conduction measurements characterized test-retest shifts in threshold. This parallelism was revealed by correlations approximating $r = +.33$ for all frequencies.

4. The inherent error of pure tone audiometry varied somewhat with frequency and the differences reached statistical significance in some instances. In particular, the reliability of threshold for 1024 c.p.s. emerged as superior in both modalities of stimulation.

5. Comparison of results obtained with two different audiometers revealed significant superiority for one instrument by both air and bone conduction. Thus, the reliability of pure tone thresholds is partially dependent upon the audiometer used.

As a final comment, two points should be re-emphasized. First, the present study did not deal with the question of threshold validity, *i.e.*, with the determination of stimulus levels required to reach normal thresholds. Rather the study was concerned with the relative reproducibility of air and bone thresholds. Second, the findings here reported contradict a commonly held opinion: namely, that the inherent error in bone conduction audiometry is woefully large compared to the error inherent in stimulation by air conduction. The present findings demonstrate that, in a clinical situation, the two modalities of stimulation can possess similar reliability. Thus, *bone conduction measurements whose precision is clinically acceptable may be anticipated whenever proper control of test conditions is maintained.*

BIBLIOGRAPHY.

1. BEASLEY, W.: Normal Hearing by Bone Conduction. National Health Survey, Hearing Study Series, Preliminary Report, Bull. 4, 1935-1936.

2. EDWARDS, A. L.: Statistical Analysis for Students in Psychology and Education. New York: Rinehart Co., 1946.

3. GREENBAUM, A.; KERRIDGE, P., and ROSS, E.: Normal Hearing by Bone Conduction. *Jour. Laryngol. and Otol.*, 54:88-92, 1939.

4. HUGHSON, W., and WESTLAKE, H.: Manual for Program Outline for Rehabilitation of Aural Casualties Both Military and Civilian. *Tran. Amer. Acad. Ophthal.*, supp., 1944.

5. LIERLE, D., and REGER, S.: Correlation Between Bone and Air Conduction Acuity Measurements Over Wide Frequency Ranges in Different Types of Hearing Impairments. THE LARYNGOSCOPE, 56:187-224, 1946.

6. MCNALLY, W.; ERICKSON, T.; SCOTT-MONCRIEFF, R. and REEVES, D.: Clinical Observations of Bone Conduction. *Jour. Laryngol. and Otol.*, 52:295-430, 1937.

7. SENTURIA, B., and THEA, A.: Bone Conduction in Audiometry: Part 1, Literature Review and Report of Preliminary Observations. THE LARYNGOSCOPE, 52:675-687, Sept., 1942.

8. WITTING, E., and HUGHSON, W.: Inherent Accuracy of a Series of Repeated Clinical Audiograms. THE LARYNGOSCOPE, 50:259-269, Mar., 1940.

TOPICAL ANESTHESIA IN ENDOSCOPY.*†

A Critical Evaluation with Case Reports and Survey of Current Opinion.

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INTRODUCTION.

The average young otolaryngologist uses topical anesthesia unconcernedly, generally keeping on with the drug and technique used during residency training. Although fully aware that a confrère occasionally runs into difficulty, he blissfully carries on confident in "his" procedure until rudely jarred by a serious reaction, or death. Having had no difficulty for possibly several hundred cases, he finally rationalizes that he is entitled to have a reaction. Judging from the material published, at some fateful time a series of accidents may befall him; he then proceeds to question his more experienced confrères and search the literature. Such was my experience and response. In an effort to determine the present concept of safe yet efficient topical anesthesia for laryngoscopy, bronchoscopy and esophagoscopy, the literature was carefully considered, and in conjunction with Dr. E. J. Chapman questionnaires were sent to the members of the American Broncho-Esophagologic Association. The findings were evaluated and my procedure was modified accordingly. A brief summary of the literature, case reports, results of the survey and my impressions are submitted herewith.

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CHEMISTRY AND PHARMACOLOGY.

Thomas and Fenton,[1] quoting Hirschfelder and Bieter (1932), divide local anesthetics into four groups:

Group I—Benzoyl derivatives, including cocaine. Cocaine is a benzoic ester of methyl ecgonine. Metycaine is also a benzoic acid ester.

Group II—Para-amino benzoyl derivatives. Pontocaine and procaine are related under this group. Procaine is an esterification of para-amino benzoic acid and diethyl-amino-ethanol. Pontocaine, butyn, larocaine and others are esterifications of para-amino benzoic acid and either aliphatic or amino alcohols.

Group III—Phenetidin derivatives.

Group IV—Quinoline derivatives.

Adriani[2] combines Groups I and II as complex esters. In summary, cocaine is a benzoic acid ester; pontocaine and procaine are para-amino benzoic acid esters. All are basic alkaloids, the contained nitrogen accounting for the basicity. They are *soluble* in lipoids, which accounts for their affinity to nerve tissue cells. The actual mode of local action beyond this is not known, except in speculation.

Factors affecting the usefulness of local anesthetic drugs are solubility, local irritation, potency and toxicity. Potency and toxicity are not necessarily parallel. They are an inherent part of the chemical structure of the drug. By a structural modification of procaine, pontocaine is derived, which is 10 times as potent[2] and, according to method of testing on animals, 10 to 25 times as toxic. Highly potent drugs as pontocaine are characterized by prolonged action. Also, the establishment of anesthesia requires a longer time interval.

Practical application of the information above, which will be discussed in more detail, is that where a known sensitivity to cocaine exists, it may be possible to use pontocaine without untoward reaction, or vice versa. Likewise, with a history of sensitivity to procaine, sensitivity to pontocaine, a chemically

related drug, should be suspected. In using a drug as potent as pontocaine, it is wise to wait five to 10 minutes after the administration of a safe dose in order to judge the intensity of anesthesia rather than risk a reaction by impatiently increasing the dose.

COMBINED USE OF TOPICAL AGENTS.

As to the value of combined use of anesthetics, Sollmann[3] states that the efficiency of mixtures of the local anesthetics corresponds to more or less complete summations, generally without potentiation. He states, further, that there would be little or no practical advantage in such mixtures. The use of pontocaine and cocaine in the same patient has been explained on the basis that the cost of pontocaine is approximately half that of cocaine in comparable concentrations. Then, why not use pontocaine altogether? This implies one of two things — either that cocaine is a more efficient anesthetic agent or that pontocaine would have to be used in relatively toxic doses to get a comparable effect.

POTENTIATION.

Regarding the use of other chemicals for potentiation of topical anesthetics, Sollmann[3] (1918) reported that potassium salts may give considerable potentiation, but that this seems to hold mainly for motor fibres and not for sensory fibres. As to potentiation by alkalinization, Sollmann affirms that sodium bicarbonate increases potency of most local anesthetics two to four times, including their use for mucous membrane application. This does not apply to pontocaine, since it is precipitated by alkalinization. The mixtures, however, do not keep well and must be made just before use [Sollman (1918) and Gwinn and Ferber[4] (1936)]. I am aware of no clinical reports evaluating alkalinization.

ADRENALIN.

The status of epinephrine in association with local anesthetics bears clarification. Gurd and Sachs[5] studied this prob-

lem, using white mice, and correlated their work with some clinical studies. The purpose of adding vasoconstrictors is twofold: first, to retard local absorption and so reduce toxicity, and second, for hemostasis which is not concerned in topical anesthesia. Several conflicting reports on the effect of epinephrine on toxicity are quoted. Gurd and Sachs found that adrenalin in dilution of 1:85,000 was without effect on cocaine toxicity, but that concentrations of 1:50,000 or stronger actually increased toxicity. They feel, then, that the doses recommended as maximum by the Mayer report[6] do not diminish toxicity; they merely have no effect upon it. No appreciable difference was found between cocaine and procaine. They point out that clinical articles making statements that adrenalin decreases toxicity without experimental backing warrant skeptical evaluation. These investigators conclude that adrenalin does not seem to decrease toxicity. It may increase it and the only advantage of adrenalin is in hemostasis, which is not relevant to topical anesthesia.

Sollmann[3] states that it is desirable and often necessary to delay absorption of local anesthetics by constricting the local circulation by adding epinephrine. He points out that the symptoms of cocaine poisoning are largely those of sympathetic stimulation and that a sensitization to epinephrine is a manifestation of major concern. Thus, in hypersensitive individuals, the nervousness induced by the adrenalin may make them more susceptible to cocaine collapse.

The following was extracted from the Mayer Report:[6]

"Cocaine is not as readily destroyed in the liver as procaine and with large doses of cocaine combined with adrenalin, the synergistic action of cocaine and epinephrine then develops to cause death. In any event, epinephrine should not be added in an amount of more than 1 mgm. (1 cc. of 1:1,000 solution)."

Dr. Harry Gold, of the Department of Pharmacology, Cornell University Medical College, reported to the Mayer Committee that one-half of the average fatal dose of cocaine for

cats becomes fatal with the addition of 0.18 mgm. of epinephrine per kilogram to the cocaine.

Fussgaenger and Schaumann[7] (1931) have shown that one drop of 1:1,000 adrenalin added to 10 cc. of 0.5 per cent solution of pontocaine allows the use of twice the lethal dose when given subcutaneously in rabbits; five drops allowed five times the lethal dose. No clinical corroboration is available.

MORPHINE.

Morphine also warrants consideration. The Mayer Committee[6] concluded that morphine has a distinct field of usefulness in allaying the apprehension of nervous patients, but at the same time sounded a warning against the administration of large doses or repeated small doses because of its synergistic action with local anesthetics on the respiratory center. They also cautioned against the routine use of morphine and adrenalin in the treatment of reactions. Sollmann[3] repeated this admonition. Unger and Thomas, in discussing Thomas and Fenton's[1] paper, agreed that morphine is contraindicated in allergic individuals.

DETOXIFICATION.

Derbes and Engelhardt[8] state that while many believe detoxification of local anesthetics takes place in the liver, Goldberg, Koster and Warshaw[9] (1943) feel that at least a fraction is rendered inert in the blood stream. Adriani[2] points out that those drugs which are eliminated unchanged in the urine or are slowly detoxified are more toxic than those drugs which, like procaine, are hydrolyzed into the original acid and alcohol which comprise their structure. Cocaine, he states, is only partially hydrolyzed. This may explain the relative toxicity of cocaine as compared to procaine, but the theory falls down with regard to pontocaine, which supposedly breaks down like procaine. Goodman and Gilman[10] (1941) point out that the liver can detoxify one minimum lethal dose of cocaine in 60 minutes. It would seem wise to proceed with caution in the face of liver damage.

IRRITANT QUALITIES.

Dietrich and Beutner[11] tested various topical anesthetics on rabbits' eyes to determine their irritant qualities. Procaine was found to be least irritating and next to procaine, cocaine. In comparing cocaine and pontocaine, the highest nonirritant concentrations were 2 per cent for cocaine and 0.1 per cent for pontocaine. As the clinical equivalent of 10 per cent cocaine is 1 or 2 per cent pontocaine, the irritant qualities of pontocaine become still more pronounced. Jackson and McReynolds[12] affirm that pontocaine has a tendency to cause irritation in patients, which may persist for several days. Schmidt[13] and Fussgaenger and Schaumann,[7] both writing in 1931, used pontocaine alone and found no irritation in the eye using 0.5 to 1 per cent in humans and 2 per cent in rabbits; however, the American manufacturer in the leaflet accompanying each bottle of 2 per cent pontocaine has this to say:

"In the eye pontocaine hydrochloride occasionally causes a transient burning sensation, usually observed when using concentrations higher than 0.5 per cent. On very rare occasions pontocaine hydrochloride, in sensitive patients, has produced lacrimation, photophobia and chemosis."

DRUG IMPURITIES.

Thomas and Fenton[1] submitted to the Committee on Pharmacology and Chemistry of the American Medical Association specimens of 2 per cent pontocaine causing reactions, and these were reported free of impurities.

EFFECT OF AGE OF PATIENT.

Shumacker,[14] working with guinea pigs in two age groups, stated that there was no great difference in the median lethal dose. He was of the opinion that reactions in the aged are due rather to the greater incidence of cardiovascular deficiences, liver damage, etc.

THE TOXIC REACTION.

A discussion of the toxic reaction to local anesthetics becomes confusing when it attempts to go beyond a clinical

description of various types of reaction. One obvious fact is that an overdose will cause reactions of varying degree up to death. Less well understood types of reaction are idiosyncrasy and allergy. Questions raised and discussed, but by no means answered are listed. In certain toxic reactions, respiratory paralysis occurs initially, with circulatory failure a late complication. In others, circulatory failure appears to be primary. One group of anesthetics is considered safer because there is a wide margin of safety between respiratory paralysis and circulatory failure. Ether and procaine are in this group.

In another group containing nupercaine (percaine) and chloroform, there is little differentiation. Pontocaine is chemically related to procaine and may be expected to fall into the safer group, and some animal studies seem to corroborate this viewpoint. Few articles give specific information based on detailed studies with regard to pontocaine. There does exist, however, a certain amount of animal study and clinical evidence which is noncorroborative.

Cocaine is believed to cause circulatory failure. Lundy and Essex[15] place cocaine as only a probable member of the more dangerous group along with nupercaine and chloroform. Sollmann, however, makes the statement that respiratory paralysis is the usual cause of cocaine deaths. According to *Veterans Administration Bulletin*, No. TB10 A-106,[16] death from cocaine poisoning is due to cardiovascular failure probably by direct action on the heart. Neither Sollmann nor the *Bulletin* give the source of study, whether on animals, etc. The question then arises: "Is pontocaine a safer drug than cocaine on the basis that it causes primarily respiratory paralysis, which has some prospect of reversibility, whereas cocaine is a more dangerous drug in that it causes primarily circulatory failure as generally believed?"

The next question is: "Is the effect purely central, purely peripheral (as direct myocardial action), or both?" Respiratory failure is commonly caused by the interference with respiration by convulsions.

According to Shumacker,[14] when guinea pigs are given a lethal dose or more of procaine by any route where absorption presumably takes place slowly (subcutaneously or intramuscularly), the sequence of events is ordinarily fairly uniform. In this order, he finds grogginess, diminution or loss of consciousness, irritative central nervous system phenomena, namely, muscular twitchings and purposeless movements, followed by generalized convulsions and finally death. Intravenous procaine, on the other hand, in the same dose gives a more rapid "collapse" reaction with or without convulsions. In Shumacker's opinion, the mechanism of death remains the same. With either route of administration, he says, death depends upon the presence in the blood stream of a lethal concentration which is roughly the same, regardless of its source. An interesting implication is that what are described by some writers as the separate "convulsive" and "collapse" types, with respiratory death in the first, and cardiac death in the second, are actually the same process, the only difference being the speed with which a lethal dose is built up. Sollmann, in discussing cocaine poisoning, implies the same clinical course for cocaine. He states that in rapid cocaine paralysis, the convulsive state, as well as the prodromal psychic phenomena, may not appear.

After recording the sequence of events with relatively slow absorption of a lethal dose of procaine in guinea pigs, Shumacker was impressed by the fact that cardiac and respiratory failure occur almost simultaneously. After further experimentation on dogs, he stated:

"The abrupt and almost simultaneous cardiac and respiratory failure suggests (rather) that death ensues from central medullary paralysis although this is not definitely proved."

He mentions the work of Knoefel *et al.*, in 1930, *1.* using intravenous cocaine on cats with the suggestion that death was due to direct paralysis of the heart, and *2.* using subcutaneous cocaine with the suggestion that death was respiratory.

Sollmann's description of cocaine poisoning roughly parallels that given for procaine in guinea pigs by Shumacker. The symptoms of cocaine poisoning, he feels, are largely those of sympathetic stimulation. He calls the reaction a sensitization to epinephrine. The effects, he points out, are mainly central, but partly peripheral. The circulatory effects are those of epinephrine poisoning with cardiac acceleration.

In describing the central actions of cocaine, Sollmann describes them as variable and complex, depending largely on dose. There is an initial stage of cerebral stimulation which is psychical and shows as alertness, loquacity, elation or anxiety and flutter. This is more marked in excitable individuals, and excitable individuals are more apt to develop reactions. This phase of initial stimulation may be absent in overwhelming cases or be only fleeting. These prodromal symptoms are often considered hysteria or apprehension by the uninitiated anesthetist.

Next comes depression characterized initially by incoordination. The author has seen this incoordination manifested as a fumbling and difficulty in holding on to the tongue. As with Shumacker's guinea pigs when given procaine, movements lose their purposive nature and become choreic. Then, as the central depression enlarges, generalized narcosis takes place. Last, epileptiform convulsions ensue and, as previously mentioned, in more severe reactions may be absent.

Fussgaenger and Schaumann,[7] working in the pharmacological laboratory of the I. G. Farben Industry, stated in their summary that death from pontocaine is always due to respiratory paralysis with an unchanged heart. In the body of their paper, however, they chart that respiratory paralysis was the cause of death in all rabbits tested with novocaine and cocaine as well.

Most of the information above is based upon animal experimentation and cannot be unconditionally applied to humans. Its practical value will evolve only when correlated with carefully observed and impartially studied clinical cases, which,

in turn, are of no value unless honestly reported in the literature. McCluskey,[17] in an article expounding the relative safety of pontocaine for spinal anesthesia, cites three cases of his own. In one instance 50 mgm. of procaine combined with 10 mgm. of pontocaine were used. Respiratory failure occurred shortly after injection without change in the blood pressure and pulse. In the remaining two cases doses of 35 mgm. and 20 mgm. of pontocaine, respectively were given. In the former, artificial respiration was used for 20 minutes until the respiration, *blood pressure and pulse returned to normal.* In the latter, circulatory failure occurred after artificial respiration for 31 hours. He then quotes Pfitzner (1931), who reported a series of 85 spinals with pontocaine. In the first five, Pfitzner used 5 to 10 mgm. of pontocaine in a 0.5 per cent solution with severe *reactions characterized by collapse, pallor, dyspnea and change in pulse rate.* In the next 80 cases, Pfitzner used 5 to 8 mgm. of pontocaine in 0.25 per cent solution without a reaction.

It would appear that Pfitzner, when using even smaller doses than McCluskey employed, had five reactions of a circulatory nature. In the remaining cases he changed the dose but slightly; however, he used a more dilute solution which, as we shall show later, slows absorption and so allows detoxification to keep the blood level down. It is also possible that the manner of anesthesia used by McCluskey, even with large doses, may have influenced the type of reaction.

Shumacker,[18] in a clinical report, cites three nonfatal procaine reactions, two characterized by circulatory collapse, but only one by sudden cessation of respiration and loss of consciousness. He concludes that one must suspect that in human beings, as in laboratory animals, the important factor is the rate of absorption and the amount of the anesthetic absorbed, and that upon these factors depend the duration and the character of the reaction.

So far, it may be said that the effect is largely central with evidence that peripheral actions can take place. On unfirm grounds, it is suggested that peripheral effects may be a func-

tion of dose and speed of absorption, or perhaps individual susceptibility. The mode of death is respiratory, cardiac, or both. It is doubtful that any drug specificity in the mode of death or type of reaction exists.[3,14] Any belief that pontocaine is safer than cocaine from the standpoint of reaction reversibility does not appear to be well founded. *Technical Bulletin* 10 A-106, Veterans Administration,[16] states that in pontocaine hydrochloride reactions, patients seldom if ever exhibit prodromal signs or symptoms of anxiety, excitement, or garrulousness. This may be due to the fact that a more toxic drug is being used and when a toxic blood level is attained, this phase may be too rapidly passed through to be noted.

The types of reaction have been classified by various authors. Reactions to overdosages are generally described as mild and severe. The severe reactions are then broken down into *1.* those with convulsions and a relatively slow course, and *2.* those without convulsions, or the rapid collapse type.

Some authorities lump idiosyncrasy and allergy under the heading of hypersensitivity. Idiosyncrasy or hyperergy (Vaughan[19]) is described as occurring in persons reacting in a normal manner but excessively, as those who sunburn easily. At any rate, reaction occurs on occasion to minute amounts of drug, or to less than the quantity needed for anesthesia.[2] Reaction is of the rapid or collapse type. The underlying mechanism is not clearly understood. Obviously, with the present state of our knowledge, little can be done about it. Fortunately, it is the least common type of reaction.

ALLERGY.

Allergic reactions undoubtedly occur. Vaughan,[19] in his "Practice of Allergy," distinguishes between hyperergy and allergy. In allergy the response is with urticaria, asthma, etc. Jackson and McReynolds[12] report asthmatic attacks and wheezing occasionally produced by pontocaine. Durbes and Engelhardt[8] state that Waldblett (1935) reports urticaria, vasomotor rhinitis and asthma from nupercaine, cocaine and

similar anesthetics. They further state that allergic shock can occur and that it may occur with what appears to be the first exposure to the drug. Allergy in that case is, then, questionable unless one considers the previous use of a chemically related drug as the sensitizing agent. They give as examples the phenolic essential amino-acids, including para-amino benzoic acid which is a component of the "B" group of vitamins with a wide distribution in nature. In reviewing case reports in the literature, there are many instances noted where reaction was obviously due to overdosage, but surprisingly there were also many cases of allergy supported by positive sensitivity tests. Thomas and Fenton[1] performed 201 patch tests using cocaine, pontocaine and controls on patients known to have allergies. Less than 0.5 per cent had plus or plus-minus reactions to cocaine. Over 9 per cent had plus or plus-minus reactions to pontocaine, and three additional patients had suggestive reactions to pontocaine. Four of these tests were later corroborated when clinical reactions to pontocaine developed. In all, some 17 cases are reported in the literature as due to pontocaine sensitivity. The majority occurred when the drug was used in the eye or were manifested as occupational dermatitis in physicians. Allergy is, of course, more obvious when manifested in these forms than as shock.

Sensitivity tests are not entirely reliable but apparently have definite value. Thomas and Fenton[1] point out that false positive and negative reactions can occur with patch tests; however, patch tests are recommended by them with the foreknowledge that they are not to be regarded as 100 per cent reliable. Dr. Paul H. Moore, in discussing their paper, stated that he believes that the markedly allergic individual should have a patch test before being referred for bronchoscopy. Thomas and Fenton[1] then report that mucous membrane tests have been used for aspirin, karaya gum, and sulfonamides. A Veterans Administration letter[20] advises that a small amount of pontocaine be applied to a restricted area of mucous membrane preliminary to its use for topical anesthesia. In the case reports will be found two cases with posi-

tive mucous membrane tests. *Bulletin* TB 10 A-106[16] recommends intradermal testing for sensitivity when indicated. Doses of 0.1 cc. of 1:1,000 solution of pontocaine, cocaine and metycaine are recommended. The patient is observed for 30 minutes for wheal formation, including redness and edema. Use of the drug giving the least reaction is recommended.

ANESTHETIC DEATH DEFINED.

With regard to anesthetic deaths in general, the Mayer Report[6] (1924) brings out the following point among those emphasized by the Therapeutic Research Committee of the American Medical Association: Dyspnea and the arrest of respiration within a few minutes after the administration of the anesthetic are especially characteristic; so much so that it is questionable whether later deaths can be charged to anesthetics.

RELATIVE TOXICITY.

Fussgaenger and Schaumann[7] give the relative toxicity based on a value of one for procaine, again on animals and varying with the route of administration, as follows:

	Procaine	Cocaine	Pontocaine
Intravenous	1	3	9
Subcutaneous	1	8	20

Clerf and his co-workers[21] have this to say with regard to pontocaine toxicity and potency:

"With the increasing employment of tetracaine hydrochloride (pontocaine) it is to be expected that toxic reactions will occur. Tetracaine is considered to possess 15 times the anesthetic power of cocaine but is two and one-half times as toxic in the same concentration."

Dr. M. H. Seevers,[22] pharmacologist, of Ann Arbor, Mich., in a symposium on anesthesia at the 1948 meeting of the American Academy of Ophthalmology and Otolaryngology, made the following points:

1. There are probably more deaths from local anesthetics than from any other single class of compounds in common use today. He asked for a show of hands on those who had witnessed a toxic reaction or a death from topical anesthesia during the past five years, and estimated that 30 to 40 per cent of the audience responded.

2. In his opinion, cocaine was better than pontocaine for pharyngeal spraying, but cautioned that only a minimum amount be used.

3. He also advised that concentrations be reduced and that the less toxic agent be used.

DILUTE OR CONCENTRATED SOLUTIONS?

Dr. Dean Lierle, in the same symposium, mentioned the advantage of the vasoconstrictive action of cocaine. He affirmed that although pontocaine produced an effect of longer duration, this was not vital in the average otolaryngologic procedure. Sollmann has this to say regarding the duration of action of local anesthetics, "The duration depends more on concentration than on the total amount of agent, so that it is more economical to use small quantities in high concentration."

As Lierle pointed out, however, long duration is not relevant. Later in the text, Sollmann makes a statement which on first glance seems contradictory unless one keeps in mind that the objectives are duration of action in the preceding statement, and rate of absorption in the following statement: "Dilution slows the absorption of cocaine and insures its thorough utilization and this economizes its use." This is an important distinction because, as found in our survey of current opinion, the majority feel that concentrated solutions of cocaine retard absorption more than dilute solutions because of their stronger vasoconstrictive action. This distinction is significant because the more dilute the solution used, the danger of toxic reaction from overdosage is by just that much reduced. There is another consideration in favor of dilute solutions which will be discussed under comments.

Shumacker found that experimental animals tolerate slightly larger amounts of procaine, if given in dilute solutions, probably because the larger amounts of the more dilute solutions are more slowly absorbed.

TOTAL SAFE DOSE.

The following recommendations are made by the Therapeutic Research Committee of the American Medical Association in the Mayer Report (1924), which is quoted by Sollmann and others: The total quantity of cocaine should not exceed 0.06 to 0.1 gm (1-1½ gr.). This amounts to 0.6 to 0.9 cc. of 10 per cent cocaine. This is not the dose recommended for actual use, but the dose which their accumulated information indicates is a safe one. It would seem, then, that the total dose which most closely approaches this figure and yet provides efficient anesthesia is the ideal one.

Sollmann, quoting Watson and Williams (1924), states that the ordinary fatal dose of cocaine is 1.2 gm. This is equivalent to 10.8 cc. of 10 per cent cocaine. *Bulletin* TB 10 A-106 of the Veterans Administration states that in effect the concentration of cocaine for anesthesia should not exceed 10 per cent (1 per cent for pontocaine) and that the total fluid employed should not exceed 10 cc. Correspondence regarding this point brought the reply that the 10 cc. solution of either cocaine or pontocaine referred to the total quantity into which pledgets were dipped and not to the quantity which might actually be absorbed by the tissues contacted. (The *Bulletin* recommends only the technique of pyriform fossa applications and states that neither drug be used in the form of a spray.) This still appears to be a dangerous statement and was so judged in our survey.

Sollmann further remarks that death has been reported from 0.02 gm., or the equivalent of 0.2 cc. of 10 per cent cocaine. This, of course, represents an idiosyncrasy. Regarding cocaine solutions, Sollmann states that they should be

freshly made and that they can safely be boiled for a few minutes. At the Mayo Clinic cocaine solutions are made up for each day's use.

The Mayer Report concluded that topical anesthesia with cocaine can be accomplished without danger by the use of carefully measured amounts of solution of low concentration and that all doses of local anesthetics, however applied, should be measured accurately.

CHOICE OF DRUG – PONTOCAINE OR COCAINE?

The question now arises, "Is there sufficient evidence to warrant making a definite choice between cocaine and pontocaine?" They are by far the most commonly used topical anesthetics in endoscopy today. I believe that cocaine is the drug of choice. Sollmann states, and I quote:

"Cocaine is satisfactory from the standpoint of efficiency, penetration and practical absence of tissue injury. Its rather high toxicity has led to the use of substitutes. Some of these present advantages in special fields, although they lack the general applicability of cocaine."

Pontocaine, although cheaper, is more toxic. Adriani,[2] as does Seevers, advised the use of the least posible amount of the weakest solution of the least toxic drug. Adriani points out that an error of 1 mgm. in estimating the dosage of pontocaine is comparable to an error of 10 to 15 mgm. of procaine. Cocaine is intermediate here. Sollmann emphasizes the fact that *a large proportion of accidents are due to mistakes in drugs, concentrations and dosage.*

Allergy to pontocaine appears to be more common than allergy to cocaine.

Pontocaine is more irritating than cocaine.

Evidence that the mechanism of reaction and death from pontocaine is respiratory only, with the implication of easier reversibility than the ascribed cardiac effect of cocaine, is not impressive.

Further evidence is gleaned from the more recent literature. Whalen,[23] in 1944, reported on the increasing number of serious pontocaine reactions. Thomas and Fenton,[1] in 1943, reported seven cases of pontocaine reaction with three deaths. They state that the use of pontocaine has been discontinued in the Cleveland Clinic.

Dr. Paul H. Moore, in the discussion of the Thomas and Fenton paper, remarked that he had used 2 per cent pontocaine excluusively in endoscopy for six years before he encountered a reaction, and this was a fatality. Within a two-month period thereafter he had two more reactions, one almost fatal. Cocaine was then used without incident, but the period of its use and the comparative number of cases were not given. Doane and Cohn[24] (1945) and Ahroon[25] (1946) report deaths from pontocaine. Two other articles and our cases reporting reactions are not admissible as both pontocaine and cocaine were used. The paper by King and Jones[26] (1948) is commendable for their faithful reporting and for the fact that they have abandoned pontocaine and now use cocaine in a 4 per cent solution for a maximum total dose of 0.16 gm. or 2.4 gr. Their technique embodying use of a relatively weak cocaine solution in a dose approaching the Mayer Report recommendation is a step in the right direction. Jackson and McReynolds[12] list several objectionable features of pontocaine and have this to say about cocaine:

"Cocaine is admittedly the most universally efficacious of the drugs used for local anesthesia and, if used with care, serious reaction will be rare."

PREOPERATIVE MEDICATION AND TREATMENT OF UNTOWARD REACTIONS.

The rôle of morphine has been discussed. In summary, it is indicated as a preoperative sedative in a relatively small dose, which outweighs any effect on respiratory depression which may occur in a later developing toxic reaction. Once a toxic reaction has occurred, it is contraindicated because of

its respiratory depression effect. Adrenalin is also contraindicated in nonallergic toxic reactions.

Barbiturates are of some value in prophylaxis of the convulsive manifestations only; they are certainly not specific. Seevers points out that anyone who believes that barbiturates are protective against serious toxicities *in prescribed doses* is in "a fool's paradise."

Shumacker, in controlled animal experiments, reports his results with coramine and metrazol in the treatment of toxic reactions. Both proved ineffective. They were administered intravenously to animals having previously received a lethal dose of procaine. For each drug the dosage given was that found to be the maximum safe dose as determined by control studies.

As the survey below will reveal, the mean preoperative routine favored is 1½ to 3 gr. of nembutal two hours before operation, and morphine, gr. one-sixth, and atropine, gr. 1/150, one hour before operation.

There are two main objectives in the treatment of an untoward reaction. One is directed toward the control of convulsive manifestations. The second is the maintenance of adequate respiratory and cardiovascular functions. The following is copied verbatim from the *Technical Bulletin* 10 A-106 of the Veterans Administration[16] and outlines the treatment of untoward reactions recommended:

"*a.* In the presence of untoward reaction exhibited by pallor, cyanosis or thready pulse, administer oxygen.

"*b.* In the presence of muscular twitchings, discontinue the instrumentation and administer oxygen by intermittent positive pressure. If the twitchings continue, administer pentothal sodium, 2 to 3 cc., intravenously, being careful to maintain a patent airway.

"*1.* Having produced muscular relaxation, continue resuscitation by means of intermittent positive pressure with

oxygen, keep needle in vein and administer additional pentothal sodium as required to control muscular spasms.

"*2.* It is futile, in the presence of frank convulsions, to inflate the patient's lungs by administration of oxygen by intermittent positive pressure on the breathing bag. The first requisite is to produce muscular relaxation by the administration of pentothal sodium which in turn will permit movement of the chest. Only sufficient pentothal sodium should be employed to control the convulsions. Oxygen may then be effectively administered if the airway is kept patent either by endotracheal tube or by tracheotomy. Do *not* administer adrenalin chloride or ephedrine sulfate in the presence of an untoward reaction to cocaine hydrochloride or pontocaine hydrochloride. The combination may produce ventricular fibrillation.

"*c.* In the presence of a continued low systolic blood pressure, administer fluids intravenously: normal saline, glucose in 5 per cent concentration, plasma or blood as indicated. Continue treatment with pentothal sodium, inhalation therapy with oxygen and administration of supportive fluids intravenously until the cocaine hydrochloride or the pontocaine hydrochloride has been detoxified. Keep the patient under observation until he recovers from the effects of the pentothal sodium."

The following equipment should always be available, and this list also is taken largely from the same *Bulletin*:[16]

1. Oxygen with mask and bag for the purpose of inflating the lungs (closed system).

2. A syringe of 20 cc. capacity containing pentothal sodium in 2.5 per cent solution.

3. Moser life-saving tubes or Magill endotracheal tubes (with metal bite) of the proper size.

4. Laryngoscope for tube insertion.

5. Tracheotomy set.

6. Pharyngeal airway, preferably of Guedel type.

Anesthesiologists generally recommend pentothal sodium for the control of convulsions. Laryngospasm may develop, however, and it is for this reason that means for maintaining a laryngeal airway must be on hand. For the same reason, when pentothal sodium alone or combined with curare is employed for general anesthesia, preliminary topical anesthesia of the larynx is recommended to minimize the complication of laryngospasm.

SURVEY OF MEMBERSHIP OF THE AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION: SALIENT FEATURES.

In July of 1948, questionnaires were sent out to the members of the Association. Sixty-five were completed. Most of the points covered in the previous review and discussion were included. Some of the salient features are listed below. In 1940, Jackson and McReynolds[12] reported a survey of the same organization and the results will be compared where feasible.

Status of preliminary pharyngeal spraying:

Forty-four felt that it should be used for reasons of efficiency and/or relative safety. Eighteen considered it unnecessary or unsafe, and of these, 13 considered it unnecessary, two unsafe, and three did not distinguish or felt that it was both unsafe and unnecessary.

Status of "cross-action" forceps or applicators with swabs applied to the pyriform sinuses as a preliminary measure:

The question was stated so as to bring out a comparison with pharyngeal and aditus laryngis spraying. Forty-one considered the pyriform technique more effective, safer or both. Twelve considered it more effective, 10 safer, and 19 both; 26 considered the pyriform technique less effective, less safe,

or both. Of these, 15 considered it less effective, three less safe, and eight both. There was some overlapping in that eight used both methods at the same sitting. The consensus was, then, 44 in favor of pharyngeal spraying and 41 for the pyriform technique. In the 1940 survey the results were approximately the same; however, in our survey only 18 considered the spray unnecessary or unsafe, whereas 26 considered the pyriform technique less effective or less safe.

Choice of drugs:

This was broken down in the questionnaire to differentiate for the spray, pyriform technique and intralaryngeal instillation.

For the spray technique, pontocaine was favored over cocaine, 29 to 19. Butyn and nupercaine received one vote each. Of the pontocaine users, the majority, 19, used the 2 per cent solution with 1 to 3 cc. as the favored amounts. One-half cc. was the least amount used and 5 cc. the most. For cocaine, the majority, five, favored the 10 per cent solution. The mean amount for the 10 per cent solution was 1.25 cc. Amounts used varied from two to three bursts for the least, to 1.5 to 5 cc. for the most.

For the pyriform technique the tables were turned, and cocaine was favored over pontocaine, 36 to 14.

For pontocaine the majority, 10, favored the 2 per cent solution with 2 cc. the favored amount. From 0.5 to 10 cc. were quoted. For cocaine the strengths varied from 4 to 20 per cent. Six favored a 5 per cent solution, eight a 10 per cent solution, and seven a 20 per cent solution. For the 5 per cent solution, amounts of from 1 to 15 cc. were given. For the 10 per cent solution the range was from 2 to 3 drops to 4 cc. For the 20 per cent solution, the range was from 0.25 cc. to 4 cc. It should be mentioned that questions called for a distinction between the usual amount used and the maximum safe amount. The answers allowed no useful tabulation on this score.

Regardless of the strength used (except for the 20 per cent cocaine users) when the technique was given the most favored was two to three applications in each pyriform sinus of one minute duration each. Only one 20 per cent cocaine user listed a technique; he used one to two applications of 20 to 30 seconds' duration each, to each side. As an extreme instance, one operator using 10 per cent solution recommends soaked applicators to be held for three minutes on each side.

Only one commented on the problem of estimating the amount of solution in contact with the tissues and so made available for absorption, and strangely enough this was in reference to the spray technique.

Intralaryngeal instillation:

Here again cocaine was favored over pontocaine, 30 to 17. For pontocaine the majority, eight, favored the 2 per cent solution with the mean amount 3.4 cc. The range was from 0.5 cc. to 10 cc. of the 2 per cent solution.

The majority of the cocaine users, 11, favored the 10 per cent solution with a mean amount of 2.4 cc. The range was from 0.5 cc. to 5 cc. One 20 per cent user recorded 5 cc. as the maximum amount used.

In the 1940 survey cocaine was also preferred; the vote was 45 for cocaine as against 17 for pontocaine. No figures were given on the basis of a breakdown according to the mode of application.

Routine questioning for sensitivity to cocaine, pontocaine, novocaine, etc.: 41 routinely questioned patients as to previous reactions and 23 did not.

Premedication routine:

The most commonly mentioned drug was the barbiturate nembutal. Only 10 did not recommend the routine use of a barbiturate. A few did not use morphine. The most favored routine was nembutal, gr. 1½-3, two hours before operation, and morphine sulfate, gr. 1/6, and atropine sulfate, gr. 1/150,

one hour before operation. In the 1940 survey morphine was the most commonly used drug and 21 did not use barbiturates.

Technique of anesthesia for esophagoscopy:

Some 40 different answers were tabulated, ranging from no anesthesia to swallowing, gargling, spraying or application with as much as 2 cc. of 20 per cent cocaine, or 6 cc. of 2 per cent pontocaine, to "GOE in 60 per cent of cases."

Combined topical anesthesia and intravenous pentothal with or without curare received occasional mention.

Status of general or intravenous anesthesia:

Fourteen did not use it. Twelve used pentothal sodium alone. Thirteen used pentothal and curare and of these, six mentioned preliminary topical anesthesia. Twelve used ether or gas-oxygen-ether. In general, it was considered indicated in foreign body work in children and in anatomically difficult or unusual cases. It is used occasionally for esophagoscopy or direct laryngoscopy and less often for bronchoscopy.

CASE REPORTS.

The following seven case reports include one death and six minor reactions, three of these exhibiting allergic manifestations. The same preoperative routine was followed for all, namely: nembutal, 1½ gr., the night before and two hours before operation, and morphine sulfate, gr. 1/6, and atropine, gr. 1/150, one hour before operation. It was also routine, dating from the third case, that one drop of 2 per cent pontocaine be placed behind the lower lip the morning of operation. The anesthetic technique was the same used at this one institution since the tenure of the author began in April, 1946; it consisted of spraying the soft palate, pharynx and aditus laryngis mainly with 2 per cent pontocaine spray. The amount used was at the time roughly gauged to be a maximum of 2 cc. in the more difficult cases.

In retrospect, I am under the impression that amounts of 4 cc. or more may have been used. This will be discussed

below under comments. Following the pontocaine spray, 1 cc. of 10 per cent cocaine was instilled into the larynx under mirror visualization using a syringe and a laryngeal cannula. Anesthesia was not carried out, or was incomplete in certain cases as noted in the specific case reports. In one instance a reaction occurred after pontocaine spray and before cocaine could be used. All cases representing toxic reactions occurred within a period of three months, the last occurring Oct. 31, 1946. Two allergic reactions to pontocaine have occurred since this date.

Case 1: Miss A. M. A. had been bronchoscoped by the same physician, April 2, 1946, without untoward incident. On Aug. 6, 1946, this patient came into the clinic with routine preparation. She was apprehensive and perturbed about possible damage to expensive bridge work. The operator was having difficulty exposing the epiglottis when the patient suddenly became rigid and exhibited mild twitchings of the lips, hands and toes. The pulse and respiration were of good quality throughout. Pupils were not dilated. No cyanosis or skin pallor developed. After several minutes all twitchings ceased and no further manipulation was attempted. No specific treatment was necessary.

Case 2: Mr. J. L. F. came in with routine preparation, Aug. 8, 1946. After routine use of pontocaine spray and before any cocaine could be instilled the patient seemed unable to grasp his tongue. He tried several times, using his right hand, and then developed a tonic spasm of the right upper extremity, followed by clonic convulsions of the face and all four extremities. There were no psychical phenomena. Respirations and pulse were of good quality, pupils undilated, and there was no cyanosis or pallor. After a minute the patient relaxed and was returned to the ward without operation or special therapy.

Case 3: Mr. N. N. P. came in for bronchoscopy, Oct. 15, 1946. He was cheerful and rational to the instant of reaction. Preoperative medication and anesthesia were routine as to amounts of drug used and technique. As the last of the cocaine was dropped between the cords, the patient had difficulty holding on to his tongue. He appeared panicky and attempted to rise from his chair, but fell back and immediately developed violent generalized convulsions. Respirations were stertorous and the pulse could not be obtained at the wrist. An attempt was made to inject sodium amytal into an antecubital vein, but the veins were collapsed and the arms in violent motion. One cc. of 1:1,000 adrenalin was injected into the heart, the patient placed on a litter and intranasal oxygen started pending the arrival of a resuscitation apparatus with positive pressure oxygen bag and mask. This arrived shortly and artificial respiration was maintained for a considerable length of time after the heart could no longer be heard to beat. Death appeared to be due to respiratory and cardiac failure. It was not determined which appeared to fail first. Autopsy showed no cause of death.

Case 4: Mr. J. S. reported to the clinic for bronchoscopy, Oct. 18, 1946. Preoperative medication and anesthesia were routine. After bronchoscopy had been completed uneventfully, the patient sat up and developed

stertorous respirations with slight cyanosis and thready pulse. Mild clonic twitchings of the extremities were noted. Sodium amytal in a dose of 3¾ gr. was slowly administered intravenously as oxygen was given by nasal catheter. Pulse, color and respirations returned to normal within a few minutes.

Case 5: Mr. L. B. O. had been previously bronchoscoped five times using the same drugs and technique. On Oct. 31, 1946, he came in and examination of the record shows no notation as to whether the mucous membrane test was positive or not. After the patient was placed on the table a macular rash was noted on the chest and upper extremities and there was slight twitching at the angles of the mouth. Pulse and respiration were of good quality throughout. Twitchings ceased on administration of sodium amytal. No operation was performed. This would appear to be a drug reaction of allergic character.

Case 6: Mr. C. C. came to the clinic in a stupor, May 6, 1947. Preoperative medication was routine. Pulse (rate 100), respirations, and skin color were normal. Both axilla were reddened and appeared mottled. The mucosa behind the lower lip was reddened. Bronchoscopy was then performed uneventfully without topical anesthesia. This is definitely a case of pontocaine allergy.

Case 7: Mr. X came into the clinic for a bronchogram. Before anesthesia was started, the mucosa behind the lower lip was not checked. After one squirt of 2 per cent pontocaine on the soft palate the patient's eyes rolled up and he appeared about to faint. The lower lip was pulled back, disclosing a markedly reddened mucosa. The patient volunteered the information that this area was "burning." Sodium amytal was given intravenously and 15 minutes later anesthesia was cautiously resumed, using cocaine alone, without incident. This, again, is without doubt a case of pontocaine allergy.

Three of the reactions are evidently due to pontocaine alone. As pontocaine and cocaine were both used in the others, the causative drug cannot be identified. Two of the reactions were due to proven pontocaine allergy and one to a drug allergy, probably pontocaine.

COMMENT.

In view of the preceding material gleaned from the literature, and in the light of the recent survey and my experience, the drugs and technique used for topical anesthesia were modified or changed. From our results, I concluded that we had an efficient anesthesia in pontocaine spray combined with cocaine intralaryngeal instillation; however, I realized that we did not have a safe anesthesia and it is in this regard that my interest in topical anesthesia was developed. My aim was to find the least amount of the most reasonably dilute solution of the least toxic drug compatible with efficient topical anesthesia for endoscopy.

From April, 1946, to March, 1949, 3,609 anesthesias for endoscopic procedures, laryngeal cauteries, bronchograms, etc., were carried out. In the first 2,715 cases we used 2 per cent pontocaine spray followed by 10 per cent cocaine (1 cc.) instillation. It had been our practice to spray the aditus laryngis until the patient could be seen to inhale and exhale spray vapor, indicating relaxation of the protective pharyngeal and aditus reflexes. Then 1 cc. of 10 per cent cocaine was instilled into the larynx fractionally in a speed depending on the tolerance of the patient. The number of spray bursts in an average case was counted and on repeating the procedure in a measuring glass I estimated that we were using a maximum of 2 cc. of 2 per cent pontocaine as a spray.

As the series of reactions developed from August to October, 1946, the spray was used more cautiously. Our patients were instructed not to swallow during the spraying, but to inhale the vapor and to expectorate at the conclusion of each series of spray bursts. Less compression on the atomizer bulb was used in order to reduce the amount of solution delivered. This served to prolong the procedure and to slow the absorption to allow detoxification to take place, and also made the most of each drop of solution. The interval between the spray and the laryngeal instillation was lengthened, usually by spraying one patient, then spraying a second patient, and then returning to the first for the laryngeal instillation.

In the meantime, a series of 299 anesthesias was run by the author using the cross-action applicators in the pyriform sinuses and followed this with the laryngeal instillation. Ten per cent cocaine was used throughout. One dram of cocaine was used to dip the cotton wrapped applicators into, the cotton was squeezed out and held in each pyriform sinus one minute for two applications. Then 1 cc. of 10 per cent cocaine was instilled into the larynx fractionally. A total of 5 cc. of 10 per cent cocaine was used, which amounts to 0.56 gm. (8.35 gr.). The safe dose as recommended by the Mayer Committee is 0.1 gm. (1.5 gr.).

This technique gave satisfactory anesthesia in the average case, but, in comparison with the spray technique, was found

wanting in several respects. In the first place, it is more disagreeable to the patient, especially the initiate. Deep pharyngeal contact is made from the start and held for one minute. It takes a considerable amount of cooperative effort on the part of the patient. Second, it is trying on the anesthetist and much vocal persuasive effort is required with a good percentage of patients. Occasionally an inured patient prefers this method because "the effect wears off sooner." Third, the total amount of drug employed is much more than in the spray technique. True, not all the drug is available for absorption, but similarly, in the spray technique, a portion of the amount used is exhaled as visible vapor and a portion expectorated. The total amount of drug used must be considered in either case. In our experience, it was necessary to instill additional anesthetic agent on the table with the pyriform technique more often than with the spray technique. Jackson and McReynolds[12] are also of the conviction that the spray is preferable for two reasons: first, it is much less disagreeable; second, it is more efficacious. The pyriform technique was, therefore, abandoned.

Improvement in the spray technique was then sought. To determine the exact amount of drug made available for absorption in the average efficiently anesthetized case was the next step. Six atomizers were used: three had new bulbs and three had bulbs which had already been in use for variable periods of time. A lay member of the clinic personnel was asked to compress the bulb evenly and count the number of squirts needed to fill a measuring glass to the two drams mark. The results are tabulated below:

Atomizer No. 1—New bulb	64 bursts
Atomizer No. 2—New bulb	86 bursts
Atomizer No. 3—New bulb	93 bursts
Atomizer No. 4—Old bulb	80 bursts
Atomizer No. 5—Old bulb	120 bursts
Atomizer No. 6—Old bulb	163 bursts

It would appear that this method of estimating the amounts of spray solution is highly inaccurate.

By trial and comparison, a 2 per cent cocaine spray followed by the instillation of 2 cc. of 5 per cent cocaine (1 cc. of 10 per cent cocaine was used initially) became the treatment of choice. In all, 594 cases were done with this technique to date. By careful attention to minutiae satisfactory relaxation of the pharyngeal and aditus laryngis reflexes in the average patient with 2 cc. of 2 per cent cocaine spray was obtained.

The amount used has been checked by filling the atomizer with a measured amount of solution; then, after each spraying, the amount remaining is accurately measured. In this way I know exactly how much cocaine has been made available for absorption in over 100 cases to date. These figures also show that adequate relaxation can be obtained with 2 cc. of 2 per cent cocaine in the average patient. The average amount was 2.3 cc. for all cases.

In looking back to the period when the series of reactions was taking place, frequently as much as 4 cc. of 2 per cent pontocaine and often a larger amount was used, for I was then less cautious.

The following details of technique are considered important. Only four to six weak bursts are directed against the posterior pharyngeal wall. The atomizer tip is then angled to 40 or 90 degrees, depending upon the position of the epiglottis with the tongue held. With mirror control, subsequent weak bursts are directed into the aditus, avoiding direct contact of the atomizer tip with the posterior surface of the epiglottis and pharynx. As the reflexes relax, the glottis comes easily into view and the spray is discontinued. In apprehensive, sensitive patients as much as 3 or 4 cc. will be needed for relaxation. The advantage of using a solution as dilute as 2 per cent is that even in refractory cases, the total amount of cocaine base is less than if solutions of 4 to 10 per cent are used. Occasionally a patient will allow direct instillation with little preliminary spraying, but the active pharyngeal reflex may make adequate exposure of the glottis difficult on the table.

We have found that 2 cc. of 5 per cent cocaine instilled into the larynx drop by drop, avoiding, especially early, direct contact of the laryngeal cannula with the epiglottis will allow at least the last 0.5 cc. of solution to be instilled with no or with a minimum of coughing or restraining effort on the part of the patient.

By means of this technique very satisfactory anesthesia with a total of 0.16 gm. (2.34 gr.) of cocaine was obtained. In the occasional case in which as much as 4 cc. of 2 per cent cocaine spray is needed, the total dose then becomes 0.2 gm. (3 gr.) of cocaine. With the pyriform technique we used 0.56 gm. An ideal atomizer for pharyngeal spraying would be one of 4 cc. capacity with 0.5 cc. markings.

I feel that I have approached the safe dose recommended by the Mayer Committee without sacrificing efficiency. The total amount of drug used, which is the determining factor in safety, has been held to a minimum by using dilute solutions and by attention to minutiae of technique, making every drop count. In spraying the pharynx and in instilling into the larynx, fractional application is followed, the slower the safer.

SUMMARY.

1. The controlling factors in the development of toxic reactions are total dosage of drug and the length of time consumed in applying that dose. Safety can best be attained by using the least amount of the most dilute solution of the least toxic drug compatible with efficient anesthesia.

2. Cocaine is the drug of choice. Pontocaine has acquired a certain popularity. It was ostensibly developed in the search for a less toxic substitute for cocaine. Pontocaine is, however, more toxic than cocaine, a more active allergen, and more locally irritating. It is cheaper than cocaine. The majority of endoscopists still rely upon cocaine for efficient action where efficiency counts, namely, for direct application to the larynx and tracheobronchial tree.

3. A procedure has been presented which in my cases most nearly approaches the ideal for topical anesthesia. It is not essentially new, but is as yet not popular through lack of appreciation of the minutiae involved.

4. Armamentarium and a procedure for handling of the toxic reaction are recorded.

BIBLIOGRAPHY.

1. THOMAS, J. W., and FENTON, M. M.: Pontocaine, Fatalities and Constitutional Reactions Following Use. *Jour. Allergy*, 14:145-159, Jan., 1943.

2. ADRIANI, J.: Some Practical Aspects of the Chemistry and Pharmacology of Local Anesthetic Drugs. *South. Med. Jour.*, 39:143-149, Feb., 1946.

3. SOLLMANN, T.: A Manual of Pharmacology. 7th ed. Philadelphia: W. B. Saunders Co., 1948, pp. 262-279.

4. GWINN, C. D., and FERBER, E. W.: Relationship of Hydrogen Ion Concentration to Efficiency and Toxicity of Local Anesthetic Solutions. *Jour. Amer. Dent. Assn.*, 23:1298-1308, July, 1936.

5. GURD, M. R., and SACHS, I.: The Effect of Adrenalin on the Toxicity of Local Anesthetics. *Quart. Jour. Pharmacy and Pharmacology*, 12:713-723, Oct.-Dec., 1939.

6. MAYER, E.: The Toxic Effects Following the Use of Local Anesthetics; an Analysis of the Reports of Four Deaths Submitted to the Committee for Study of the Effects of Local Anesthetics of the American Medical Association and the Recommendations of the Committee. *Jour. A. M. A.*, 82:876-885, Mar. 15, 1924.

7. FUSSGAENGER, R., and SCHAUMANN, O.: Ueber ein neues Lokalanaesthetikum der Novokainreihe (Pantokain). *Arch. f. exper. Path. u. Pharmakol.*, 160:53-65, June, 1931.

8. DERBES, V. J., and ENGELHARDT, J.: Deaths Following Use of Local Anesthetics in Transcricoid Therapy; Critical Evaluation. *Jour. Lab. and Clin. Med.*, 29:478-482, May, 1944.

9. GOLDBERG, A.; KOSTER, H., and WARSHAW, R.: Fate of Procaine in Human Body After Subarachnoid Injection. *Arch. Surg.*, 46:49-58, Jan., 1943.

10. GOODMAN, L., and GILMAN, A.: The Pharmacological Basis of Therapeutics. New York: The Macmillan Co., 1941.

11. DIETRICH, W. C., and BEUTNER, R.: A Search for the Least Irritant Topical Anesthetic. *Anesthesiol.*, 7:255-259, May, 1946.

12. JACKSON, C. L., and MCREYNOLDS, G. S., JR.: Anesthesia for Peroral Endoscopy. *Ann. Otol., Rhinol. and Laryngol.*, 49:1048-1053, Dec., 1940.

13. SCHMIDT, H.: Ein neues Lokalanaesthetikum der Novocainreihe (Pantocain). *Chir.*, 3:97-104, Feb., 1931.

14. Shumacker, H. B., Jr.: Reactions to Local Anesthetic Agents: I—Experimental Studies with Procaine. *Surg.*, 10:119-133, July, 1941.

15. Lundy, J. S., and Essex, H. E.: Experiments with Anesthetics: III—A New Local Anesthetic Pontocaine, for Prolonged Anesthesia. *Laboratory and Clinical Observations. Proc. Staff Meeting, Mayo Clin.*, 6:376-380, June 24, 1931.

16. Veterans Administration: *Tech. Bull.*, 10 A-106, Jan. 16, 1948.

17. McCluskey, C. F.: Pontocaine as a Spinal Anesthetic. *Anesthesia and Analgesia*, 12:116-118, May-June, 1933.

18. Shumacker, H. B., Jr.: Reactions to Local Anesthetic Agents. II—A Clinical Report. *Surg.*, 10:134-144, July, 1941.

19. Vaughan, W. T., and Black, J. H.: Practice of Allergy. 2nd ed. St. Louis: C. V. Mosby Co., 1948, p. 40.

20. Veterans Administration: Letter from Medical Director, July 6, 1942: Caution in Use of Butyn Sulfate and of Pontocaine.

21. Clerf, L. M.; Spencer, J. T., Jr., and Riggs, C. D., Captain, M.C., U.S.N.: Progress in Otolaryngology. Peroral Endoscopy. *Arch. Otolaryngol.*, 47:356, Mar., 1948.

22. Seevers, M. H.: Symposium: Anesthesia in Otolaryngologic Surgery. *Tran. Amer. Acad. Ophthal. and Otolaryngol.*, pp. 281-322, Mar.-Apr., 1949.

23. Whalen, E. J.: Anesthesia in Peroral Endoscopy. *Ann. Otol., Rhinol. and Laryngol.*, 53:469-471, Sept., 1944.

24. Doane, J. C., and Cohn, E. M.: The Toxic Effect of Pontocaine Employed Locally. *Anesthesiol.*, 6:421-423, July, 1945.

25. Ahroon, W. A.: Report of a Death from Pontocaine Hydrochloride. The Laryngoscope, 56:320-323, June, 1946.

26. King, J. W., and Jones, D. H.: Anesthesia in Endoscopy. The Laryngoscope, 58:1195-1200, Nov., 1948.

AUREOMYCIN IN PENICILLIN-RESISTANT ACUTE OTITIS MEDIA.

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The etiological organism of acute otitis media is almost always sensitive to penicillin. The rarity of surgical mastoiditis or chronic purulent otitis media in recent years is evidence of this. Because penicillin is effective, most men incise the tympanic membrane and follow it with intramuscular penicillin routinely; however, on occasion one sees a case in which penicillin is not effective. In such a case, culture of the discharge should be made and sensitivity to the various antibiotics determined. In those cases in which the organism is sensitive to aureomycin, it should be given.

CASE REPORT.

This patient, a 15-year-old white girl, reported on March 19 with an acute otitis media. A myringotomy was done and 300,000 units of procaine penicillin with 2 per cent aluminum monostearate was given intramuscularly. This was followed by similar doses of pencillin on March 21, 22, 23, 25, 28, 29 and 31. On each of these days the ear was examined and pulsation of the drum was seen. The discharge remained profuse. On April 1, the laboratory* reported that culture from discharge collected two days before showed hemolytic staphylococcus aureus (mannitol and coagulase positive), and that it was resistant to greater than four-tenths of a unit of penicillin per cc., but was sensitive to less than 1 microgram of aureomycin per cc. April 2, the patient began to take aureomycin, one dose of 500 mg. followed at four-hour intervals with doses of 250 mg. of aureomycin. (Patient weighs 122 pounds, or 55 kilo.). The next day the same procedure was followed. April 4, she was examined and discharge was still profuse, pulsation of the drum still present. The dose was then increased to 500 mg. every four hours. On the following day, April 5, there was a decrease in discharge, and on April 6 the canal showed very slight moisture, and there was no pulsation of the drum.

*Thanks are due Dr. C. E. McLeod for the laboratory work.

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Due to the nausea, medication was discontinued for three days. When the moisture persisted, aureomycin, 250 mg., was given every four hours and when examined after 48 hours the canal was absolutely dry.

DISCUSSION.

Before the days of penicillin and the sulfonamides, an acute otitis media which was going on to resolution would drain for from 10 days to three weeks; however, the pulsation of the drum, which is a sign of the severity of the inflammatory lesion, would subside in the first three or four days and the discharge would gradually but definitely lessen after the first week. In this patient at the end of two weeks there were no signs of abatement, and within three days after the beginning of aureomycin therapy (a low dosage the first two days), the lesion resolved itself. Considering the above, I believe the aureomycin was specific and not coincidental with a spontaneous cure.

COMMENT.

Since the number of cases of otitis media that are resistant to penicillin are few, I feel justified in reporting one case. Culture from cases clinically resistant to penicillin, determination of sensitivity to aureomycin and specific treatment of aureomycin-sensitive cases should corroborate my findings.

EPICEREBELLAR SUBDURAL HYGROMA.*†

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The name "subdural hygroma" was suggested in 1932 by W. Dandy to emphasize the basic feature common to all subdural collections of fluids without regard to their etiology.

The first observation of such a subdural lesion was made by C. H. Mayo in 1894. He saw a 12-year-old boy who had hemiplegia and stupor after a head injury. Mayo called the released subdural fluid "braincyst." In 1922, E. Payr gave a classical description of observations made during the first World War. He stated that a sudden traumatic rupture of the arachnoid is the most common cause for the development of collections in the subdural space. He also found subdural collections of fluid in head injuries without a rent in the arachnoid. He observed three soldiers with tangential head wounds which presented adjacent to the area of the scalp injury, subdural secretions which Payr called "meningitis serosa circumscripta traumatica." He believed that the traumatic pressure from outside was the main factor responsible for the development of those subdural lesions.

Streit proved by his experiments that lateral pressure leads to the same reaction in primary inflammatory lesions such as an extradural abscess or lateral sinus thrombosis after mastoiditis. The subdural space has poor capacity for absorbing transudates or exudates. It does, however, resemble the pleural and peritoneal cavities in the readiness with which it

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forms adhesions, the "iron curtain" against invaders. These features are also expressed by designations such as protective or sympathetic pachymeningitis circumscripta (H. Brunner).

Naftziger was the first American neurosurgeon who published his experiences in subdural collections of fluids (1924). Similar publications followed by Ira Cohen, Walsh, Sheldon, M. Scott, DaCosta, Adson and Wycis. During the second World War, Adam McConnell and his group, in England, reported on their war observations. This study included the largest number of cases observed by any group of examiners. In 1945, H. Wycis, giving an account on seven personal observations, made a detailed analysis of the 99 cases reported so far in the literature. In 33 cases he was able to enumerate the clinical symptoms observed. Severe headache was the most common complaint, occurring twice as often as all the other symptoms: dizziness, irritability, loss of memory, restlessness, aphasia, hemiparesis and hemiplegia, as well as drowsiness; semicoma and stupor were also noted.

It is interesting that Ménière's symptoms were not prominent in the reported cases. The following two cases observed at the Jewish Hospital are, therefore, of particular interest.

Case 1: A. S., a female, white, 39 years old, was admitted on Oct. 10, 1946, to the medical ward for observation because of dizziness. The patient was discharged on Oct. 18, 1946, after a general check-up had not revealed evidence of an organic lesion. She was readmitted five weeks later, on Nov. 25, 1946, to Dr. M. Scott's neurosurgical service with the diagnosis "Ménière's disease." Her *chief complaint* at that time was vertigo, nausea, vomiting, buzzing in left ear, sensation of pressure in occiput and occasional slight headache. This began five months before admission, with dizziness and "funny" feeling in her occiput. Severe vomiting began two weeks before readmission and dizziness and nausea were worse whenever she turned her head to the left.

Physical Examination: Tenderness of left occiput to pressure; slightly swaying gait to the left; spontaneous horizontal nystagmus to left; decreased corneal reflex, left; tuning fork lateralized to left ear; mild degree of hearing loss, left (10 per cent); slightly increased patellar reflex, left; finger-nose test poor, left; slight adiadochokinesis, left; laboratory and Roentgen studies were negative.

A cerebellar tumor was suspected and a *suboccipital craniotomy* was performed on Nov. 29, 1946 (surgeon: Dr. M. Scott). The exposed left cerebellar dura appeared markedly distended. An incision made in the dura released one and one-half ounces of clear fluid. No evidence of a cerebellar or cerebellopontine angle tumor was found. Biopsy of cerebellar tissue revealed degenerative changes of the Purkinje's cells.

The *postoperative course* was satisfactory. The patient complained of blurred vision for two weeks after the operation; fundoscopic examination, however, was negative. She was discharged from the hospital on Dec. 24, 1946, four weeks after the operation, in good condition. The preoperative symptoms of dizziness, adiodochokinesis and swaying gait had disappeared.

Comment: A middle-aged woman developed Ménière-like symptoms over a period of five months. At the time of her first admission no organic changes could be found. On readmission, five weeks later, definite neurological signs indicating intracranial pressure upon the brain stem, including the left hemicerebellum, were observed. A suboccipital craniotomy was performed and a subdural hygroma of the left cerebellar hemisphere was released. The patient made a good recovery.

The etiologic basis for this hygroma was not demonstrated. No history of trauma or mastoid infection could be obtained. This, then may be an example of Dandy's idiopathic subdural hygroma due to a local imbalance of proper secretion and resorption of the cerebrospinal fluid.

Case 2: J. L., white female, 36 years old, married nurse, was admitted to the writer's service on Aug. 25, 1948. She was in the Valley Forge Army Hospital for eight months under observation and discharged from service in November, 1947, with the diagnosis of "Ménière's syndrome and migraine." Her present illness began three years before, in 1945, with episodes of dizziness, nausea and loss of balance. Spells returned; these attacks lasted a short time and were preceded or accompanied by excruciating headaches radiating from the occiput to the left temple and the left eyeball. For the past three months the condition had become worse. Her weight decreased 30 pounds and she suffered from severe insomnia.

In 1937, her left mastoid was operated upon at Women's College Hospital because of chronic ear infection, the onset of which started 13 years previously. A week after the mastoid surgery, the jugular vein was ligated. Drainage from left ear stopped and the mastoid wound closed.

Physical examination revealed a thin, pale, irritable but cooperative patient. There was a well healed left mastoid scar and slight degree of combined deafness, left. Tuning fork was lateralized to left. Bone conduction was good. There was a normal excitability of both labyrinths. Spontaneous rotary horizontal nystagmus to left (first degree) could be observed. Gait was unsteady, swaying to the left. Romberg was positive; patient fell to the left. Turning head to right, left, backward or forward did not change the tendency of swaying to the same (left) side. There were typical adiadochokinesis, left, slightly increased triceps and patellar reflexes, left, slight hypesthesia of the entire left side, and no abdominal skin reflexes, left. Laboratory tests revealed increased sedimentation rate (27 and 31, respectively), otherwise negative findings. Spinal punc-

ture showed normal pressure, clear fluid, normal chemistry. Pneumoencephalogram demonstrated minimal degree of dilatation of the cortical subarachnoid pathways.

A peripheral inflammatory cerebellar lesion was suspected and on Sept. 7, 1948, I performed a postauricular left craniotomy. Bone covering cerebellum and temporal lobe was very sclerotic and hemorrhagic. The dura was markedly distended and reddened. Sigmoid sinus was collapsed. A large net of distended veins covered the cerebellar and temporal dura. A vein crossing Trautmann's triangle was thrombosed. The thrombus was removed and in front of this area the dura was punctured. About 8 cc. of turbid xanthochromic fluid were withdrawn, until the dura collapsed and presented regular pulsation.

The fluid withdrawn at operation did not contain any organisms. Histological investigation revealed polymorphonuclear leucocytes, occasional lymphocytes and fibrin.

Postoperative course was rather stormy; headaches persisted for the first three weeks, but they lost the pounding and boring character. For the first week the patient complained of air hunger and weakness of the left leg. Three blood transfusions were given. In the fourth week she developed a purpura of the lower legs. Prothrombin and capillary fragility (Rumpel and Leed) tests were negative. Minimal degree of jaundice was present. The patient was discharged from the hospital on Oct. 2, 1948, four weeks after the operation. She was seen by the writer in his office for the next two months and was discharged in February, 1949. Her gait was normal again, dizziness had disappeared, occasional slight headaches recurred, area of exposed dura was still tender to pressure at the time of her last visit. Patient was advised to return after several months for a check-up and a possible plastic repair of the postauricular tender areas.

Comment: Here, also, a middle-aged woman presented Ménière-like symptoms. The history, however, was more complicated. A left-sided chronically discharging ear had to be operated upon 11 years ago (1937). A week after mastoidectomy the thrombosed sigmoid sinus was exposed and the jugular vein ligated. Eight years later (1945), Ménièriform attacks with severe hemicrania occurred. They recurred more often the first year (1946) and incapacitated the patient for work.

At the time of admission, the patient presented definite clinical findings characterized by incoordination of posture and motility. Romberg test revealed a tendency to fall to the left; the gait was swaying to the left; adiadochokinesis, hypermetria and dysmetria were present on the left side. Rotatory-horizontal nystagmus to the left of first degree could be observed. These phenomena proved at least a topographical diagnosis; namely, a physiopathology of the left hemicerebellum.

Which anatomical changes were responsible for these hypofunctional reactions of the left cerebellum was our next problem. There were several possible lesions: *1.* Degenerative, due to impaired blood supply; *2.* space taking: *a.* arachnoiditis adhesive serosa, *b.* frontal lobe tumor, *c.* extracerebellar inflammatory lesion.

1. Our first thought focused to possible degenerative changes of the cerebellar centers due to lack of proper blood supply. The otological literature recites a number of examples where cerebral accidents occurred after the ligature of the jugular vein in sinus thrombosis (Linser, Bruns, Stolz, Ruttin, Urbantschitsch). M. Ersner and D. Myers called attention especially to the vulnerability of the left sinus and the left jugular vein; they found that in 75 per cent of their observations on patients, the diameter was much smaller than that of the right side.

In the beginning we were inclined to favor a degenerative etiology because of the negative laboratory findings. The severe headache, however, increased tendon reflexes and hypesthesia of the left side pointed to a space taking lesion which could be *2.* arachnoiditis adhesive serosa (Oppenheim, Pette, Placzek and Krause) of the cisterna lateralis, a condition observed and described by Bárány in several cases after extradural abscess and lateral sinus thrombosis, in tuberculosis and syphilis. In some textbooks this specific arachnoiditis is called "Bárány's symptom complex." Recently L. Fleischmann reported a classical case observed years after a healed radical mastoidectomy. It resembles the clinical manifestations of a pontocerebellar tumor. The common feature is complete deafness, and a nonreactive homolateral labyrinth is seen in both; a choked disc and a high spinal pressure are often seen in "Bárány's disease." Those phenomena were not observed in our patient.

3. A tumor of the right frontal lobe could elicit pseudo-Ménière syndromes as seen in our patient. The frontal lobe sends impulses to the contralateral cerebellar centers via the frontopontine system and the middle cerebellar peduncle. A

tumor can impair those impulses and consequently the cerebellum dependent on them (E. A. Spiegel). Roentgen studies, including pneumoencephalogram, excluded neoplasms. Thus "per exclusionem," our conclusion was

4. An extracerebellar inflammatory lesion. I contacted Dr. Emily L. VanLoon, who did the mastoid operation in 1937; she gave me valuable information on her observations made at that time. She informed me that a thrombosis of the lower portion of the sigmoid sinus was found; that the septic temperature dropped to normal after the ligature of the jugular vein; that the excruciating headaches of which the patient had complained before the operation were still noticed a long time after the patient was discharged from the hospital. This report gave rise to a new question: "Was the sinus thrombosis the only otitic complication at that time?" A meningitis serosa often occurs simultaneously with sinus thrombosis, especially in children and young adults (H. Brunner). L. Hayman could prove the incidence of a pachymeningitis interna after sinus thrombosis had been provoked in experimental studies. The meningitis serosa may resolve spontaneously, may localize with formation of adhesions (pachymeningitis serosa interna circumscripta) or may progress, causing a subdural empyema (Preysing). It is possible that the infection went through all these stages in our case. The turbid fluid withdrawn from the subdural space contained many leucocytes, but no organisms. Apparently immune biological forces had in time overcome the virulence of the infection, had absorbed a portion of the cystic content and left behind what we may call another variety of a "subdural hygroma." While this is probably the sequence of events, it is not possible to exclude the independent occurrence of this lesion seven to eight years later, in 1945, when the patient's severe complaints began. A dura exposed by a previous mastoid operation can be very sensitive to reinfection, especially in the presence of marked sclerosis of the temporal bone, indicative of recurrent otitis media.

SUMMARY.

1. A historical review of "subdural hygroma" is given.

2. Two cases of epicerebellar subdural hygroma presenting Ménière-like syndromes are reported. The etiology of the first case is unknown; it is presumably an "idiopathic hygroma" (Dandy). In the other case the hygroma developed apparently from a previous subdural empyema as a sequence of severe mastid infection.

3. Both patients recovered.

4. No reports of Ménièriform syndromes due to subdural hygroma were found in the reviewed literature.

REFERENCES.

1. Adson, A. W.: See DaCosta, D. G.

2. Barany, R.: *Verhandl. d. Deutsch. Otol Gesellschaft*, 108, 1912.

3. Brunner, H.: Intracranial Complications of Ear, Nose and Throat. Chicago: Year Book Publishers, 1946.

4. Bruns, P., and Linser, P.: *Beit. z. klin. Chir.*, 28:642, 1900.

5. Cohen, Ira: *Arch. Neurol. and Pysch.*, 18:709, 1927.

6. DaCosta, D. G., and Adson, A. W.: *Arch. Surg.*, 43:559, 1941.

7. Dandy, W. E.: In Lewis: Practice of Surgery, XII:291, 1948; in Kopetzky: Surgery of the Nose, Nelson & Sons, 1947, p. 387.

8. Ersner, M. S., and Meyers, D.: The Laryngoscope, 43:800, 1933.

9. Fleischmann, L.: *Acta Otolaryngol.*, 36:17, 1948.

10. Haymann, L.: *Arch. f. Ohrenh.*, 83:1, 1910.

11. Linser, P.: See Bruns, P.

12. Mayo, C. H.: *N. Y. Med. Jour.*, 59:434, 1894.

13. McConnell, A. A., *et al.*: *Jour. Neurol. and Psych.*, 4:237, 1941.

14. Naftziger, H. G.: *Jour. A. M. A.*, 82:1751, 1924.

15. Oppenheim, H., and Borchardt, M.: *Deutsch. med. Wchnschr.*, 36:57, 1910.

16. Payr, E.: *Med. Klin.*, 12:84, 1916.

17. Pette: *Ztrlbl. f. Neurochir.*, 112:86, 1936.

18. Placzek and Krause: *Berlin. klin. Wchnschr.*, 44:94, 1907.

19. Preysing: Cited by Bayer, H., in Operationen am Ohr, C. Kabitsch, Leipzig, 1931.

20. Ruttin, E.: *Monatsschr. f. Ohrenh.*, 53:535, 1919; 59:177, 1925.

21. Scott, M.: *Amer. Jour. Surg.*, 55:534, 1942; Surg., 13:152, 1943.

22. Spiegel, E. A.: Neurology of the Eye, Ear, Nose and Throat. Grune and Stratton, 1944.

23. Stolz: *Korrespondenzblatt der Schweizer Arzte*, No. 13, 1918.

24. Streit, H.: *Arch. f. Ohrenh.*, 83:202, 1910; 89:117, 1916.

25. Urbantschitsch, E.: *Monatsschr. f. Ohrenh.*, 52:284, 1918; 53:534, 1919.

26. Walsh, M. N., and Sheldon, C. H.: *Proc. Mayo Clin.*, 12:134, 1937.

27. Wycis, H.: *Jour. Neurosurg.*, 2:340, 1945.

RATIONAL TREATMENT OF TRACHEAL FOREIGN BODY WITH CASE REPORT.*

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Frequently, in the field of endoscopy the question of diagnosis is perplexing, while the treatment may be obvious. There are times when the diagnosis is rather obvious and the treatment a bit perplexing. Metallic foreign bodies lend themselves to adequate radiographic visualization. The diagnosis, in these cases, is rather obvious.

The incidence of open safety pins in the tracheobronchial tree is extremely low. Norris, in a recent report of 250 foreign bodies covering a period of five years at Chevalier Jackson Bronchoscopic Clinic, reports one open safety pin in a child of two years. The incidence of other pointed metallic objects is also low as compared with the vegetal and nonmetallic foreign bodies.

The delay in diagnosis or even suspicion of a foreign body is greater with the nonobstructing type. It will vary from a few hours to many years. As in the case to be reported, symptoms are intermittent and may lead to a diagnosis of severe respiratory infection or pneumonia and treatment be instituted. Once suspicion of a foreign body is aroused or a chance X-ray demonstrates it, urgency and emergency ensues. The parent can always recall that the child was playing with the same object or something similar only a few hours before.

CASE REPORT.

L. C., a 20-month-old white female, was admitted to St. Louis Children's Hospital about 10 P.M., Feb. 14, 1949, with a history of apparent health until two days before, when she developed fever, cough, irritability and anorexia. The mother recalled seeing the child playing with a safety pin on the day of the onset of the symptoms. The cough did not persist, but

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the other symptoms did until admission. During the afternoon of admission, the child was taken to her family physician and diagnosed as having pneumonia and given 300,000 units of procaine penicillin in sesame oil intramuscularly. This same diagnosis had been made on two or three occasions before and the mother was dissatisfied this time. The child had then been taken to the station hospital, Scott Field, Ill., where chest X-ray demonstrated the presence of an open safety pin in the neck. She was then referred to this hospital for treatment.

Family history was noncontributory. Past history was noncontributory except that the child had had measles and whooping cough at the age of six months, followed by "virus pneumonia" every one to two months since that time, treated by intramuscular penicillin. At the age of six months the child lost her voice suddenly one night. This was followed by crying and facial grimaces. Aphonia persisted for two days, when the voice gradually returned to a weaker and more hoarse type than previously.

Physical examination was essentially negative except for coarse breath sounds with moist rales throughout. Fluoroscopy showed an open safety pin minus the keeper, points up, in the trachea. There was slight haziness in the right upper lobe. The mediastinum was in the midline.

X-ray Report: "An open safety pin is visualized lying in the trachea at the level of the first thoracic vertebra." Admission laboratory work showed a white count of 19,200 with a moderate shift to the left. Urine was entirely negative. Examination of the vocal cords by direct laryngoscopy revealed redness and thickening of the posterior ends of the cords and the interarytenoid space. The airway was adequate, and the foreign body was not visualized.

Because of the position of the foreign body, the age of the child and the fact that two points had to be brought through the glottic chink, it was deemed advisable to do a tracheotomy and remove the foreign body through the tracheotomy wound under local 1 per cent procaine anesthesia and three-fourths of a grain of sodium phenobarbital intramuscularly. Upon opening the trachea, no evidence of the foreign body was seen. A 5 mm. bronchoscope was then passed through the tracheotomy wound to the region of the carina without the foreign body being seen. The direction of the bronchoscope was then reversed and the spring of the pin was identified just below the glottis. This was grasped and removed without difficulty, points trailing. A No. 2 tracheotomy tube was inserted; the wound was partially closed with silk sutures to the skin, and the patient was returned to the ward in good condition.

Twelve hours later, the tracheotomy tube was blocked off without evidence of respiratory distress. The tube was removed the following day. Secondary closure of the wound was made two days later. The child received a total dosage of crystalline penicillin intramuscularly, 240,000 units daily, and streptomycin subcutaneously, 600,000 units daily, for a period of seven days. The admission temperature was 37.3° C. It rose to 38.5° C. 12 hours following the removal of the foreign body and then returned to normal, where it has remained ever since.

Direct laryngoscopy by means of a Flagg laryngoscope was carried out on the fourth postoperative day. The cords were of normal color, the thickening and redness had subsided, but there was still some thickening in the interarytenoid area. The voice continued to improve and by the time the child was discharged from the hospital, on Feb. 24, the voice was of normal timbre. Physical examination was entirely normal; the wound was healing well and no sequelae were anticipated.

DISCUSSION.

In this case we were confronted with an open safety pin, point up, minus the keeper. The problem of removing a double pointed object through the larynx brought to mind many possible complications. The logical instrument seemed to be a safety pin closer in order to draw the points safely into the bronchoscope. The lack of a keeper made the disengaging of the pin with its subsequent loss a possibility. We felt that the larynx would admit a 4 mm. bronchoscope and several minutes might be required to accomplish the maneuver of bringing the pin safely into the scope. This would have had to be done with great care to prevent damage to the cricoid cartilage, with its possible resultant stricture formation. Damage to the vocal cords was to be avoided. Safety pins are also quite elusive. In attempting to remove them in the tracheobronchial tree, one frequently hears or reads of them going from one bronchus to the other.

In this case, the point seemed embedded in the region below the interarytenoid space and well fixed. Radiographic studies demonstrated it extending from the fifth to the seventh cervical vertebrae. It seemed logical to remove this through a tracheotomy opening as the most expedient and safest method.

An elective tracheotomy is a simple, safe procedure. The resulting scar is negligible. A potentially dangerous foreign body from the standpoint of manipulative trauma, damage to the cricoid or vocal cords, certainly lends itself to a simple, safe tracheotomy with removal of the foreign body through the tracheotomy wound.

Radiographic studies can be, and often are, misleading. Size and position of the foreign body can be easily distorted by the positioning of the tube in relation to the part to be examined and the film. The film demonstrated a rather large safety pin, lying low in the trachea. We expected to encounter the pin in the tracheotomy opening. When the pin was not seen in the wound, a 5 mm. bronchoscope was passed to the region of the carina without visualization of the foreign body. It was then directed toward the larynx, the pin visualized

and removed with the points trailing. The pin was small in size, as compared with the expected size from the film. It was corroded to the point of marked weakness in several areas.

Upon seeing the pin, the mother noted that it was not the pin the child had played with two days prior to admission. We feel safe in assuming that this foreign body was present some 14 months. Perhaps the keeper had separated because of corrosion and pressure and may have been coughed out. The appearance of the vocal cords, the history of hoarseness with return to normal voice following removal of the foreign body seems to substantiate this belief.

In retrospect, it is felt that an attempt at removal by bronchoscopy would have resulted in the fragmenting of the pin with subsequent lodging of portions in the smaller bronchii.

SUMMARY.

1. A case of double-pointed foreign body (open safety pin minus keeper) of trachea is presented with removal through tracheotomy wound.

2. A significant delay in diagnosis is postulated.

3. Radiographic studies may be misleading unless perfectly spotted.

REFERENCES.

1. Norris, Charles M.: Foreign Bodies in the Air and Food Passages; a Series of 250 cases. *Ann. Otol., Rhinol. and Laryngol.*, 57:1049-1071, 1948.

2. Clerf, Louis H.: Foreign Bodies in the Air and Food Passages; Observation on End-Results in a Series of 950 Cases. *Surg., Gynec. and Obst.*, 70:328-339, 1940.

3. Jackson, C., and Jackson, C. L.: Disease of the Air and Food Passages of Foreign Body Origin. New York: W. B. Saunders Co., 1936.

4. Jackson, C. L.: Endoscopy for Foreign Body. *Ann. Otol., Rhinol. and Laryngol.*, 45:644-654, 1936.

5. Jackson, C. L.: Bones as Overlooked Foreign Bodies in the Lung. *Arch. Otolaryngol.*, 12:499-507, 1930.

6. Jackson, C., and Jackson, C. L.: Foreign Bodies in the Air and Food Passages, Roentgenologically Considered. New York: Paul B. Hueber, Inc., Ann. Roentgen, Vol. 16, 1934.

3720 Washington.

ABSCESSES OF THE RETROPHARYNGEAL SPACES IN ADULTS.*†

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Abscesses of the retropharyngeal spaces occurring in infants and children have literally been reported by the hundreds. In 1909, Vas[1] described 1,054 such abscesses in patients at Children's Hospital in Budapest. In contrast to the incidence in infants and children, their relatively infrequent occurrence in adults is apparent from a review of the literature. Watson,[2] in 1903, presented one in an adult that he had seen in his years of practice and was able to find in the literature only 16 previously reported for adults.

To date, a review of the literature reveals that 114 abscesses of the retropharyngeal spaces in adults have appeared in medical reports.

To these, I am adding six gathered from the records of Barnes Hospital in St. Louis, Mo., from January, 1932, to January, 1943. In addition, I am including two I saw recently, both occurring within a period of six weeks.

Faier[3] reported seven adults with abscesses in the retropharyngeal spaces in 81,986 admissions to Barnes Hospital from December, 1914, to January, 1932, or once in 11,712 admissions. The present series of six occurred in 69,956 adult admissions to Barnes Hospital from January, 1932, to January, 1943, or one in 11,659 adult admissions.

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The rarity of this condition in adults as indicated by the paucity of clinical reports is not consistent with the records at Barnes Hospital, since 13 of the 122 cases occurred in this hospital alone.

HISTORICAL.

Mackenzie[4] cites an abscess of the retropharyngeal space reported in the second century, A. D., by Galen with spontaneous rupture. No mention was made whether or not this occurred in an adult.

The first to be reported in an adult was by Syme,[5] in 1826. This abscess occurred in a 35-year-old male and was probably the result of cervical caries. The patient recovered when a large sequestrum was removed from the pharyngeal wound.

ANATOMY.

The writings of Grodinsky,[6-8] Holyoke,[8] Weintraub,[9-10] Iglauer[11-13] and Proetz[14] have done much to refute the original idea that the area behind the pharynx was one space. Some present-day texts persist in this viewpoint.[84-85]

It would be redundant to recite in detail the anatomy of the retropharyngeal spaces as described by the above authors. A review of the anatomy of this area reveals four potential spaces behind the pharynx (see Fig. 1). The following is a brief account of these spaces:

Peripharyngeal Spaces: These are paired and intramural, separated in the midline by a vertical, fibrous septum. These spaces extend laterally to the neurovascular bundle of the neck with the internal carotid artery as the most medial constituent. They extend above to the upper margin of the superior constrictor muscle, and below, the spaces terminate behind the cricoid cartilage.

Postvisceral Space: This potential space is limited behind by the perimysium of the prevertebral muscles and anteriorly by the fascial layer investing the peripharyngeal space; the postvisceral space extends laterally to the neurovascular bun-

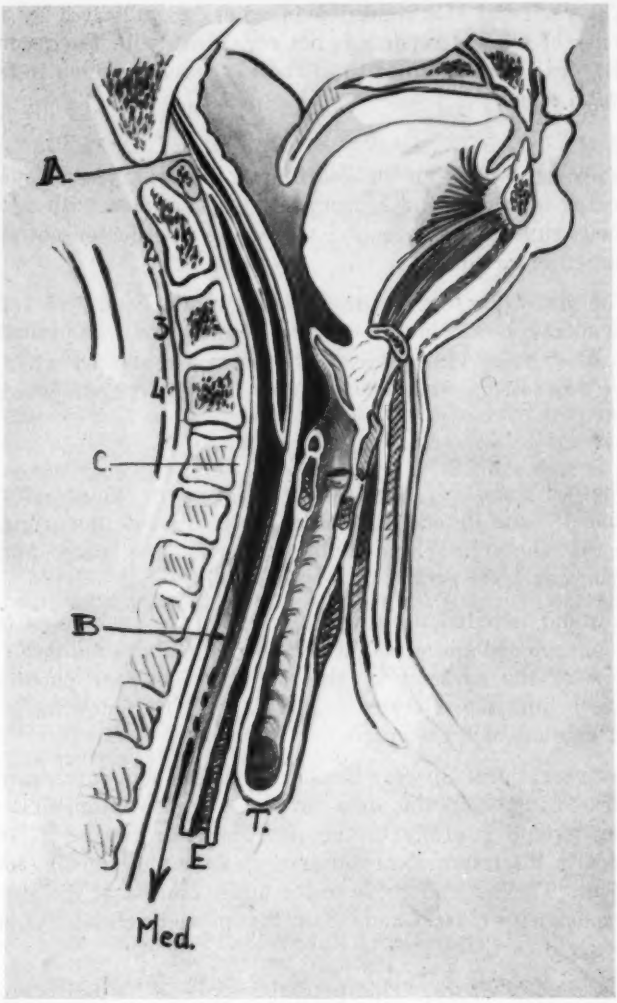

Fig. 1. Diagrammatic view* of midsagittal section of the head and neck illustrating retropharyngeal spaces. (A) Peripharyngeal space. (B) Post-visceral space. (C) Prevertebral space.

*Drawing made by Dr. Jose Bello, of Buenos Aires, Argentina.

dle of the neck; superiorly, it ends medially at the tubercle of the arch of the atlas; below, it proceeds through the superior aperture of the thorax behind the esophagus into the posterior mediastinum.

Prevertebral Space: This potential space exists behind the prevertebral fascia extending from the base of the skull to the coccyx.

Iglauer and Weintraub have recommended the elimination of the term "retropharyngeal." They prefer that the space involved be so designated instead of grouping all of the spaces together.

Grodinsky and Holyoke[8] stress the obvious importance of a thorough knowledge of the fascia and fascial spaces in anticipating the routes of spread of infection and in planning proper incisions for drainage.

Weille,[15] however, feels that splitting the cervical fascia into subdivisions has resulted in overlooking the remarkable ability of the cervical fascia to produce walling off in the presence of any inflammatory stimulus.

Lymph nodes are found adherent to the outer coat of the pharynx on each side and situated between the cupola of the peripharyngeal space and the internal carotid artery. Most[16] has indicated that these nodes drain the nose, sinuses, nasopharynx, Eustachian tube, middle ear, mastoids, pharynx, tonsils, teeth and larynx.

Thomson[17] states that these nodes atrophy after the fifth year. Frank[18] and Goldstein[19] believe the atrophy of these nodes accounts for the uncommon occurrence of abscesses in adults.

Dense, thick, fibrous bands separate the peripharyngeal from the parapharyngeal space, and the tendency for abscesses in the retropharyngeal spaces to gravitate downward may account for the fact that spread of infection from the peripharyngeal space to the parapharyngeal space has never been reported.

PATHOLOGY.

A simple peripharyngeal abscess, Iglauer[12] writes, results from the breakdown of infected lateral peripharyngeal lymph nodes which are in intimate topographic relation with the upper portion of the peripharyngeal space. Infection from these nodes would tend to extend most likely into the peripharyngeal space. From a study of the cases reviewed, one is unable to gather any definite statistics regarding the incidence of involvement of the spaces behind the pharynx. It is assumed that the peripharyngeal space is most often involved. This statement is made, however, because of the greater accessibility of the peripharyngeal nodes (Iglauer[11]) to infection and injury.

Involvement of the prevertebral space occurs most frequently with involvement of the cervical vertebrae from tuberculosis, injury with osteomyelitis, and syphilis.

Grodinsky[7] states that abscesses behind the prevertebral fascia usually gravitate to lower levels along the muscles taking origin from the vertebral column, and he states, further, that there is very little tendency for infection in this space to perforate through the prevertebral fascia into the postvisceral space.

Weintraub[10] and Grodinsky[7] have indicated that infection can spread from one space to another and with spread into the postvisceral space will result in invasion of the thorax with mediastinitis.

Furstenberg[20] suggests that mediastinitis results from direct extension along fascial planes of the neck or by lymphogenous metastases.

From a review of the literature for retropharyngeal abscesses in adults it appears that mediastinitis occurs infrequently as an accompaniment of disease in these spaces. In only 10 instances were these abscesses complicated by medastinitis.

It has been stated that abscesses in the retropharyngeal spaces may result in spread to the axilla and lateral chest, yet this complication is not noted in the review.

In the formation of otogenic pharyngeal abscesses, it is probable that the same disease process that causes the otitis media and mastoiditis results in the destruction and rupture of the peripharyngeal glands with resultant abscess formation.

McKenzie,[21] Cunningham[22] and Faier[3] have enumerated possible methods by which otogenic pharyngeal abscesses may be produced.

McKenzie:

1. By direct extension of purulent disease from middle ear through the petrous bone to the pharynx.
2. By wandering of an extradural abscess of the middle cranial fossa to the tip of the petrous bone and then by the foramen lacerum to the extracranial inferior surface of that bone.
3. By direct extension of purulent disease from pneumatic cells in the tympanomastoid osseous structures to the under surface of the occiput and then to the pharynx.
4. By wandering of an extradural abscess of the postcranial fossa to the suboccipital region.

Cunningham:

1. Infection of a retropharyngeal lymph node.
2. Direct extension forward of a middle ear suppuration.
3. Direct extension from a necrotic area in the mastoid process.
4. Exit of an extradural abscess from the cranial cavity.
5. Secondary burrowing of a Bezold's abscess.

He concludes that in retropharyngeal abscess the ear warrants investigation in every case.

Faier:

1. Propagation of infection by continuity of lesions of bone, labyrinthine cellulitis, etc.
2. Preformed nerve and vessel channels, and embryonic structures.
3. Lymphatic pathways.
4. Venous pathways.

It is likely that infection is introduced when an abscess is the result of a foreign body. The extent of involvement depends upon the depth of penetration of the foreign body.

Abscesses of the retropharyngeal spaces in the cases reviewed occurred in 49 males and 23 females. The reason for this unusual frequency in males is unknown.

ETIOLOGY.

Some confusion exists as to the most common etiologic factor in abscesses of the retropharyngeal spaces in adults.

Rimer[23] believes that most abscesses in the retropharyngeal spaces are due to acute upper respiratory infection. Smith[24] feels that influenza with severe nasopharyngitis and rhinitis is the most common cause.

Christierson[25] states that tuberculosis of the cervical vertebrae is the most frequently encountered cause.

While several texts indicate foreign bodies as a common cause, Tucker[26] has indicated that a foreign body complicated by an abscess in the retropharyngeal spaces is rare.

According to Barbour,[27] abscesses rarely occur in adults because of the immunity acquired from frequent submarginal infections which children have not acquired.

In an unusual case reported by Kistler,[28] the patient developed an abscess after a self-attempted passage of a stomach tube to relieve esophageal stenosis.

In another unusual case reported by Laszlo,[29] a patient developed an abscess after an injury to the back of his neck with fracture of the third cervical vertebra, resulting in osteomyelitis of that vertebra.

Sheedy[30] lists abscesses of the sphenoidal sinuses as one of the causes of abscesses of the retropharyngeal spaces; however, the literature makes no mention of this condition as a predisposing factor.

For the total number of abscesses in the retropharyngeal spaces reported in the literature, 72 are attributed to some etiological factor.

Causative factors of abscesses in the retropharyngeal spaces:

Upper respiratory infection	21	(37; 60; 41; 67; 74; 71; 20; 25—3 cases; 3—2 cases; 53; 7; 46; 82; 83; 3 Barnes Hospital; 1 author)
Otitis and mastoiditis	28	(23; 78; 76; 22—7 cases; 18—2 cases; 39—2 cases; 70; 62; 80; 65—8 cases; 3 Barnes Hospital)
Foreign bodies	7	(7—2 cases; 25; 26; 56; 36—2 cases)
Tuberculosis of the spine	6	(25; 38; 52; 40; 64; 1 author)
Tonsillectomy	4	(77; 75; 18—2 cases)
Syphilis	2	(18; 12)
Trauma to pharyngeal wall	1	(28)
Injury to neck with osteomyelitis	1	(29)
Dental infection	1	(3)
External otitis with parotitis	1	(3)
Unknown	49	(18—5 cases; 68—2 cases; 47; 66 (15—6 cases; 2; 35; 63—6 cases; 61; 56; 79—2 cases; 60; 59; 5; 45; 44; 43; 42; 24—7 cases; 81—4 cases; 32; 31—2 cases; 39)

According to Faier,[3] suppurative otitis media is frequently overlooked as an etiologic factor in abscesses of the retro-

pharyngeal spaces and may be of major consequence in the ultimate recovery of the patient if the association is recognized early.

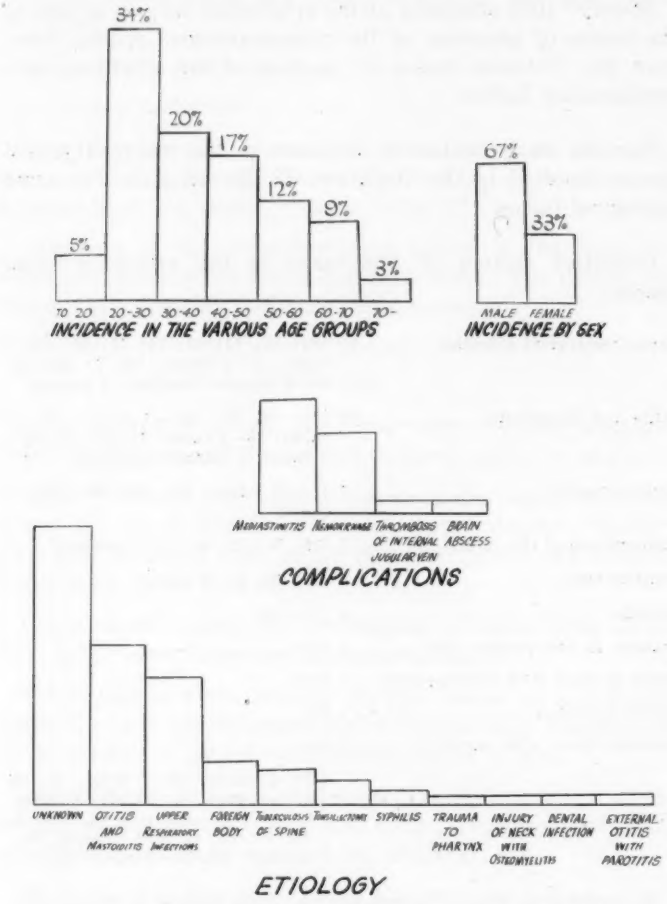

Fig. 2.

SYMPTOMS.

The following symptoms occurred in order of frequency in the cases cited from the literature:

Dysphagia
Pain in the throat
Dyspnea
Stiffness of the neck
Difficulty in opening mouth
Loss of weight
Nasal obstruction
Pain in the head, back and arms
Pain in the ear
Hoarseness
Cough

In addition to the above mentioned symptoms, almost all presented signs of toxicity with fever, chills and prostration.

DIAGNOSIS.

The diagnosis of abscess of the retropharyngeal spaces in adults is usually made quite readily, particularly in acute conditions. The history, examination of the patient, palpation of the mass, aspiration of the abscess with a needle and syringe and X-ray studies serve to reveal the true nature of the lesion. In the chronic form the abscess may be months in formation before sufficient symptoms occur to require medical attention. In one instance of the present series six weeks ensued between the original complaints about the neck and development of sufficient symptoms and signs to require attention.

The value of X-ray examination cannot be overemphasized. Barlow[31] and Kistler[28] stress the value of X-ray diagnosis. Weille[15] states that lateral X-rays of the neck will make it possible to determine the presence of retroesophageal mediastinal extension.

Woodward[32] makes it routine in his work to take lateral X-rays of the neck in all cases referred for Roentgen examination of the respiratory or upper gastrointestinal tracts and on several occasions unsuspected retropharyngeal abscesses have been discovered.

Large or small abscesses occur in a large number of cases of vertebral tuberculosis according to Sever.[33] It is his opinion that the increase in size of these abscesses represents increasing vertebral destruction. He feels that any perivertebral shadow as shown in the Roentgenogram, even without erosion in early or known tuberculosis, should be called an abscess, since this shadow represents bone detritus plus local inflammatory reaction and is, therefore, pathologically an abscess.

It has been stated by Kyle[34] that abscesses in the retropharyngeal spaces must be differentiated from aneurysms in adults. To my knowledge, the literature is devoid of any instance in which an aneurysm was found or was opened by mistake. Examination for pulsation, palpation and aspiration should reveal the presence or absence of an aneurysm.

Downie[35] reported a case of retropharyngeal swelling, with X-ray findings, simulating an abscess that was due to callus following injury to the cervical vertebrae. He states, "There had apparently been a fracture of the upper cervical vertebrae and the resulting callus formation is seen as the large, hard swelling."

In differentiating the space or spaces of the neck involved, a small amount of iodized oil injected after incision and drainage should be most helpful in determining the actual space involved. This method would also give some aid in prognosis and further therapy. In one instance a small amount of oil was injected through the incision, revealing the site of the abscess to be in the peripharyngeal space (see Fig. 4).

COMPLICATIONS.

The most common complications of abscess in the retropharyngeal spaces are mediastinitis, and hemorrhage from

involvement of one or more of the larger vessels in the neck. Adams,[36] Taylor,[37] Cline,[38] Lyman,[39] Christierson,[25] Furstenberg,[20] and Weille and DeBlois[15] have presented cases complicated by mediastinal extension; Grodinsky[7] has reported three instances of this complication.

Wylie and Wingrave,[40] Travers,[41] Chassaignac,[42] Graigie[43] Liang,[44] Tacey[45] and Insly[46] have reported complications by massive hemorrhage. Travers' patient required ligation of both the internal jugular vein and the common carotid artery.

Mosher[47-49] has brought out the importance of suspecting a phlebitis or thrombosis of the internal jugular vein, if an abscess of the retropharyngeal spaces is associated with chills or septic temperature.

In their excellent work, Salinger and Pearlman[50] point out that recurrent hemorrhage in the presence of definite swelling of the neck, for which retropharyngeal abscess has been diagnosed, indicates involvement of a major vessel and that no time should be lost in performing ligation before a final and fatal hemorrhage ensues. They state, further, that when successive hemorrhages occur from a retropharyngeal abscess, whether moderate or severe, one cannot dismiss the probability of involvement of the internal carotid artery as the most likely source of bleeding.

In an unusual case presented by Christierson,[25] the patient developed a mediastinal abscess with rupture into a bronchus.

According to Kanavel,[51] cold abscesses secondary to tuberculous disease of the cervical vertebrae may cause serious and widespread involvement because of the tendency to gravitate downward into the posterior mediastinum.

Moore[52] reports a case opened along the posterior border of the sternocleidomastoid muscle which resulted in paresis of the cervical sympathetic nerves, and Witchell[53] reported a case complicated by a brain abscess.

TREATMENT.

In the treatment of abscess of the retropharyngeal spaces in adults, some controversy exists as to the proper method of surgical approach.

Dean[54] used the external approach and wrote that not only should every case of retropharyngeal abscess which is secondary to caries of the vertebrae be drained externally, but every case of retropharyngeal abscess should be drained in this way. Dean advised a vertical incision parallel to the anterior border of the sternocleidomastoid muscle, with finger dissection in the deeper structures until the abscess is reached. Lillienthal[55] advised making the incision posterior to the sternocleidomastoid muscle and he used an aspirating needle to indicate the site of the abscess before opening.

Kanavel,[51] Smith[24] and Christierson[25] agree that all abscesses as the result of tuberculous caries of the cervical vertebrae should be drained by the external approach, and Kanavel further states that opening tuberculous abscesses through the mouth is a serious technical error, and that the lateral approach should be performed in such a manner as to prevent secondary infection.

Burckhardt[56] has reported the persistence of a draining fistula following drainage of an abscess by the external route.

Thomson,[17] on the other hand, reserves the external incision for those abscesses too low in position to be reached through the mouth, when spasm of the masseter muscles cannot be overcome, when a large pulsating vessel is noted in front of the abscess and when the abscess is more lateral.

In tuberculous involvement of the cervical vertebrae, fixation of the cervical spine is essential to healing according to Christierson.[25] Blair[57] informs me that opening an abscess due to tuberculous caries of the cervical vertebrae without adequate fixation of the head and neck may result in serious injury or death, since, in many cases, the abscess acts as a

splinting mechanism. The sudden release of this splinting may result in loss of vertebral support with spinal cord paralysis and death.

In the series of cases reviewed, 31 were opened through an oral incision and 13 were drained by the external approach. Nine of the abscesses ruptured and drained spontaneously.

Dixon[55] was the first to advise the use of two suction pumps simultaneously in suctioning off the pus after incision of an abscess through the mouth. In Case 1 this procedure was undoubtedly of life-saving value.

As regards the treatment of otogenic pharyngeal abscesses, McKenzie[21] advocated cleaning out the diseased mastoid and opening the pharyngeal abscess through the mouth.

It is felt that the use of penicillin and streptomycin will play a considerable rôle in these infections. In three of the cases presented, penicillin was used; prompt recovery followed draining of the abscesses. Streptomycin was used in two of my cases with beneficial results in one; in the other case the patient was moribund when the drug was instituted.

It is felt that abscesses of the peripharyngeal space can be adequately treated with incision and drainage through the mouth.

Abscesses of the postvisceral space should be approached externally so that the mediastinum may be adequately drained. Abscesses of the prevertebral space can be best treated by the external approach to the posterior pharyngeal structures after splinting has been accomplished.

SUMMARY.

1. Abscesses of the retropharyngeal spaces in adults occur more frequently than reports in the medical literature indicate. At Barnes Hospital in St. Louis, Mo., during a 29-year period, one case of abscess in approximately 11,700 adult admissions was seen.

2. The term "retropharyngeal" should be used only when speaking of all the spaces behind the pharynx. Whenever possible, the exact location of the abscess should be indicated. The instillation of iodized oil through the pharyngeal incision will frequently help in indicating the extent and site of involvement.

3. Abscesses of the retropharyngeal spaces in adults occur more than twice as often in males. The reason is unknown.

4. These abscesses occur most frequently following upper respiratory infections and following infection in the middle ear and mastoid.

5. The symptoms most frequently encountered are: Dysphagia, pain in the throat, dyspnea, stiffness of the neck and difficulty in opening the mouth.

6. Mediastinitis and massive hemorrhage are the most common complications of this condition. Mediastinitis occurred in 10, and massive hemorrhage in seven of the cases reviewed.

7. The use of two suction pumps simultaneously in opening abscesses of the mouth is considered essential.

Case 1 (see Figs. 3, 4, 5): A 23-year-old Negro housewife was admitted to the Homer G. Phillips Hospital on Dec. 6, 1948, with complaints of severe pain in the throat, dysphagia, dyspnea, loss of weight, chills and fever. Three weeks before admission, during an acute upper respiratory infection, she developed a sore throat with chills and fever which gradually became worse. A week before admission she developed difficulty in swallowing, and on admission was able to swallow only liquids. In attempting to swallow, she frequently returned much of the liquids through her nose. Four days before entry, she developed increasing difficulty in breathing and was forced to sleep in a sitting position. There was no history of tuberculosis, trauma or foreign body.

She has had frequent "head colds" during the winter with occasional sore throats. The left side of her face and neck was severely burned in a fire in 1934.

The physical examination revealed the patient to be well developed and fairly well nourished. She was in marked distress. She spoke with much difficulty and the mouth and pharynx were examined with considerable difficulty because of pain associated with opening the mouth. Breathing was difficult and she was unable to move the head or neck without pain.

Fig. 3. Lateral view of the neck showing massive soft tissue swelling.

The tonsils were red and small. The posterior pharyngeal wall was markedly reddened and presented a mass protruding forward and filling most of the oropharynx. On the right side, the mass was in contact with the base of the tongue. Digital examination revealed the swelling to be smooth, soft and nonpulsating. The temperature was 102° F. The nasal mucosa was reddened but no pus was seen. The ears were within normal limits. The left neck presented well healed burn scars and the right neck presented small, tender nodes in the posterior and anterior chains.

The patient was sent for immediate X-rays of the neck and chest. Medical consultation revealed the heart and lungs to be within normal limits.

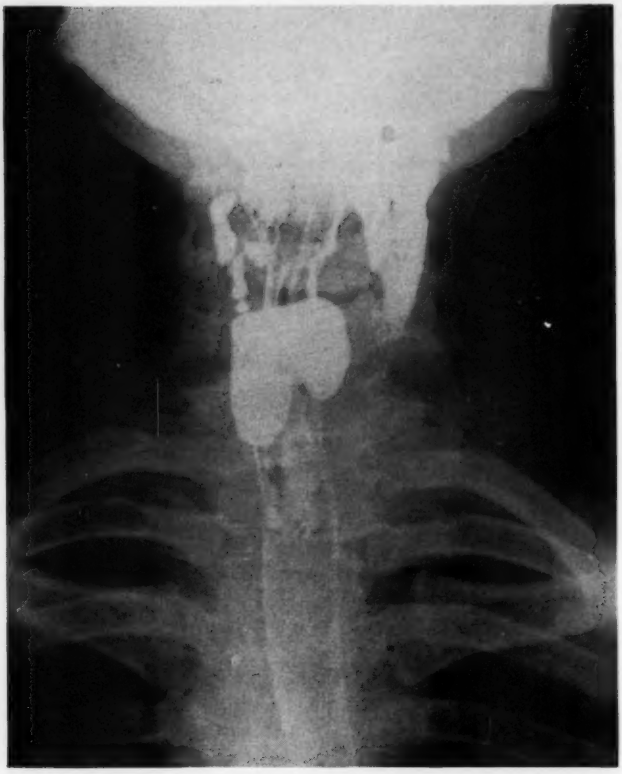

Fig. 4. Shows neck after injection of iodized oil through the pharyngeal incision. The peripharyngeal space is outlined and destruction of the vertical fibrous septum is seen.

X-ray Report (see Fig. 3): There is no evidence of destruction involving the bodies of the vertebrae and the intervertebral discs are intact. There is pronounced increase in the soft tissue distance between the bodies of the cervical vertebrae and the trachea due to soft tissue swelling. Chest X-rays were within normal limits.

Laboratory Report: White blood count, 21,000, with 60 segmented neutrophiles, 12 stab and Juvenile cells, 22 lymphocytes, six monocytes; red blood count, 3,700,000 with 80 per cent hemoglobin; urinalysis revealed 2+ albumin. The Kahn test was negative and the nonprotein nitrogen was 61.

Fig. 5. Lateral view of the neck after injection of iodized oil.

Course in the Hospital: Because of marked dehydration, the patient was given 1,000 cc. of 5 per cent glucose in normal saline intravenously and 1,000 cc. of Amigen intravenously. Penicillin in the dose of 50,000 units was given every three hours. A tracheotomy set was placed at the bedside.

The morning following admission the patient's respiratory difficulty had increased markedly. She was taken to the operating room at once. Anesthesia consisted of three or four puffs of a 10 per cent cocaine spray to the pharyngeal wall. With the patient in deep Trendelenburg position, an incision was made in the area of swelling slightly to the right of the midline on the posterior pharyngeal wall. There was an immediate gush

of thick green-yellow pus. With two suction machines in operation it was possible to keep up with the flow of pus. At one point when one suction tip became occluded, the pharynx filled with pus and respirations ceased; it was only when the other suction tip was placed in operation again that the pharynx was opened sufficiently to permit respirations again. Approximately 400 cc. of pus were recovered.

Following surgery, the patient's respiratory difficulty ceased immediately and she was able to swallow liquids with only little difficulty.

Cultures taken at surgery revealed B. pyocyaneous. The patient was placed on 0.5 gm. streptomycin twice daily in addition to the penicillin.

Fig. 6. Lateral view of the neck showing soft tissue swelling and destruction of the vertebrae.

Recovery was uneventful. The incision in the pharynx was reopened daily for seven days, at the end of which time there was no further discharge. The patient was discharged on the fourteenth postoperative day.

An examination three months postoperatively revealed the patient to have been asymptomatic since discharge from the hospital, and examination of the pharynx revealed it to be normal.

Case 2 (see Fig. 6): A 54-year-old Negro male entered Homer G. Phillips Hospital on Oct. 29, 1948. Diagnosis of Pott's disease of the right sacroiliac region was made.

X-rays at time of admission revealed destructive processes involving the right sacroiliac region and chronic pulmonary inflammatory process.

Treatment was instituted at once, but the patient's progress was unsatisfactory and his course was gradually down hill.

On Dec. 9, 1948, the patient complained of pain in the right side of the neck and an X-ray of the cervical spine is reported as revealing "a destructive process involving the greater portion of the body of the third vertebra. The anterior portion of the body was quite ragged and irregular on both its inferior and superior surfaces. There is no evidence of new bone formation. There is some bony debris in the soft tissues in the region of this body anteriorly. There is also some anterior soft tissue swelling."

On Jan. 18, 1949, the patient complained of pain in the throat and some difficulty in swallowing. Examination at that time revealed a small, red, slightly tender mass protruding from the posterior pharyngeal wall on the right side.

On Jan. 19, 1949, an ear, nose and throat consultation was requested. Examination revealed a well developed but poorly nourished Negro male in considerable distress and in an extreme state. The teeth were poor; the tonsils were red and small; a large red mass was seen on the right posterior pharyngeal wall, almost filling the entire oropharynx on that side.

X-ray examination of the cervical region of the spine revealed increasing destruction of the body of the third cervical vertebra and there was seen a pronounced swelling of the soft tissues anterior to the cervical vertebrae.

The following day the patient's condition was worse; he was having marked respiratory difficulty; lassitude and weakness had increased. The patient was obviously in no condition for extensive surgery for drainage of the abscess in his neck. Simple incision and drainage through the mouth was decided upon. The patient's neck was immobilized in a plaster cast prior to surgery.

With the patient in deep Trendelenburg position and using 1 per cent novocaine infiltration anesthesia, the mass in the pharynx was incised. Approximately 100 cc. of thin yellow pus were recovered. Two suction machines were utilized to clean the pharynx and to maintain an airway.

Cultures taken from the pus obtained at surgery were reported as revealing no growth.

Immediately following surgery, the patient was more comfortable and was able to swallow fluids with less difficulty. Postural drainage was instituted and a nurse was in constant attendance with suction to maintain a clean airway.

The patient's general condition, however, continued to deteriorate following surgery. He became increasingly more lethargic the following day and expired in coma two days postoperatively. Permission for autopsy could not be obtained.

BIBLIOGRAPHY.

1. Vas, J.: Quoted by Frank.[18]

2. Watson, J. A.: Retropharyngeal Abscess in Adults. *Northwestern Lancet* 23:192-194, 1903.

3. Faier, S. Z.: Retropharyngeal Abscess of Otitic Origin. *Ann. Otol., Rhinol. and Laryngol.*, 42:408-437, 1933.

4. MacKenzie, Morrell: Diseases of the Throat and Nose. Philadelphia: P. Blakiston, Son & Co., 1884, p. 70.

5. Syme, James: Case of Recovery After Extensive Exfoliation of the Vertebra Dentata. *Edinburgh Med. Jour.*, 25:311-313, 1826.

6. Grodinsky, M.: Ludwig's Angina, Retropharyngeal Abscess. *Jour. A. M. A.*, 114:18-22, Jan., 1940.

7. Grodinsky, M.: Retropharyngeal and Lateral Pharyngeal Abscesses. An Anatomic and Clinical Study. *Ann. Surg.*, 110:177-199, 1939.

8. Grodinsky, M., and Holyoke, F. A.: The Fasciae and Fascial Spaces of the Head, Neck and Adjacent Regions. *Amer. Jour. Anat.*, 63:367-407, Nov., 1938.

9. Weintraub, J. D.: A New and Functional Systematization of the Neck Connective Tissues. *Tran. Amer. Laryngol. Assn.*, pp. 81-109, 1939.

10. Weintraub, J. D.: A New Anatomic and Functional Systematization of the Connective Tissues of the Neck. *Arch. Otolaryngol.*, 33:1-30, Jan., 1941.

11. Iglauer, S.: Anatomic-Pathologic Studies of Retropharyngeal (Peripharyngeal) Abscesses. *Tran. Amer. Laryngol. Assn.*, 61:66-80, 1939.

12. Iglauer, S.: Anatomic-Pathologic Studies of Retropharyngeal (Peripharyngeal) Abscesses. *Arch. Otolaryngol.*, 33:31-43, Jan., 1941.

13. Iglauer, S.: Autopsy Studies of a Case of Tuberculous Peripharyngeal Abscess. *Tran. Amer. Laryngol., Rhinol. and Otol. Soc.*, 48:204-209, 1942.

14. Proetz, A. W.: In discussion of Iglauer's paper.[12]

15. Weille, F. L., and DeBlois, E.: Retropharyngeal Abscess, Problems in Treatment. *Arch. Otolaryngol.*, 39:344-347, Apr., 1944.

16. Most, A.: Zur Topographie und Aetiologie des Retropharyngealen Drusenabscessen. *Arch. f. Klin. Chir.*, 61:615-627, 1900.

17. THOMSON, ST. CLAIR: Diseases of the Nose and Throat. New York: D. Appleton & Co., 1926, pp. 464-468.

18. FRANK, I.: Retropharyngeal Abscess. *Jour. A. M. A.*, 77:517, 1921.

19. GOLDSTEIN M. A.: Retropharyngeal Abscess. THE LARYNGOSCOPE, 18:46-56, 1908.

20. FURSTENBERG, A. C.: Acute Mediastinal Suppuraton. *Tran. Amer. Laryngol., Rhinol. and Otol. Soc.*, pp. 210-229, 1929.

21. MCKENZIE, D.: Otogenic Pharyngeal Abscess. *Jour. Laryngol., Rhinol. and Otol.*, 30:12-29, 1915.

22. CUNNINGHAM, H. M.: Retropharyngeal Abscess of Otitic Origin. *Tran. Amer. Laryngol., Rhinol. and Otol. Soc.*, 33:542-557, 1927.

23. RIMER, F. H.: The Diagnosis and Treatment of Retropharyngeal Abscess. *Pa. Med. Jour.*, 42:1197-1198, July, 1939.

24. SMITH, J. E.: Retropharyngeal Abscess with Reference to Abnormally Large Percentage of Adult Cases. *Ann. Otol., Rhinol. and Laryngol.*, 49:490-499, 1940.

25. CHRISTIERSON, S. V.: Retropharyngeal Abscess in Adults with Special Emphasis on Its Etiology. *Tran. Amer. Laryngol., Rhinol. and Otol. Soc.*, pp. 133-153, 1933.

26. TUCKER, G.: Retropharyngeal Abscess from Lodgement of Foreign Body in Esophagus. *Jour. A. M. A.*, 84:511-512, Feb., 1925.

27. BARBOUR, P. C.: Retropharyngeal Abscess. *Med. Times*, New York, 67:404-406, Sept., 1939.

28. KISTLER, W. K.: Retropharyngeal Abscess, Diagnosis and Treatment. THE LARYNGOSCOPE, 41:568-576, 1931.

29. LASZLO, A. F.: Retropharyngeal Abscess Due to External Injury of Neck. *Ann. Otol., Rhinol. and Laryngol.*, 44:226-232, 1935.

30. SHEEDY, B. D.: Pharyngeal Abscess. *Med. Rec.*, 81:1226-1228, 1912.

31. BARLOW, R. A.: Retropharyngeal Abscess. *Tran. Amer. Laryngol., Rhinol. and Otol. Soc.*, 35:316-320, 1929.

32. WOODWARD, F. D.: Retropharyngeal Abscess. *Arch. Otolaryngol.*, 14:467-471, 1931.

33. SEVER, J. W.: Abscesses in Tuberculosis of the Spine. *Jour. A. M. A.*, 92:1822-1824, June, 1929.

34. KYLE, D. B.: Diseases of the Nose and Throat. New York: W. B. Saunders & Co., 1915, p. 603.

35. DOWNIE, W.: *Glasgow Med. Jour.* 1903.

36. ADAMS, J.: Two Cases of Postpharyngeal Abscess from Impaction of Fish Bones. *Lancet*, 2:156-157, 1847.

37. TAYLOR, J.: Case of Laryngitis, Pharyngeal Abscess Extending Throughout the Posterior Mediastinum from Pharynx to Diaphragm. *Lancet*, 1:462-465, 1846.

38. CLINE, L. C.: Retropharyngeal Abscess. THE LARYNGOSCOPE, 13:668, Sept., 1903.

39. LYMAN, H. W.: In discussion of Frank's paper.[18]

40. WYLIE, A., and WINGRAVE, V. H. W.: Lancet, 1:1042, 1906.

41. TRAVERS, F. T.: Retropharyngeal Abscess, Secondary Hemorrhage, Ligature of the Internal Jugular Vein and Common Carotid Artery, with Recovery. *Brit. Med. Jour.*, 2:703-704, Sept., 1902.

42. CHASSAIGNAC: *Deutsche. Ztschr. f. Chir.*, 57:591, 1902 (quoted by Salinger and Pearlman[50]).

43. CRAIGIE, D.: *N. Y. Med. Jour.*, 48:233, 1888 (quoted by Salinger and Pearlman[50]).

44. LIANG, P. T.; LIANG, P. K., and LEI, C. H.: *Brit. Med. Jour.*, 1:967, 1925 (quoted by Salinger and Pearlman[50]).

45. TACEY: *Glasgow Med. Jour.*, 1:405, 1868 (quoted by Salinger and Pearlman[50]).

46. INSLEY, J. L.: A Case of Hemorrhage from an Acute Retropharyngeal Abscess. *Jour. Laryngol. and Otol.*, 59:428-430, Nov., 1944.

47. MOSHER, H. P.: Thrombosis of Internal Jugular Vein with Pyemia as a Complication of Retropharyngeal Abscess. THE LARYNGOSCOPE, 29: 638, 1919.

48. MOSHER, H. P.: Deep Cervical Abscess and Thrombosis of the Internal Jugular Vein. *Tran. Amer. Laryngol., Rhinol. and Otol. Soc.*, pp. 326-340, 1920.

49. MOSHER, H. P.: Deep Cervical Abscess and Thrombosis of the Internal Jugular Vein. THE LARYNGOSCOPE, 30:365-375, 1920.

50. SALINGER, S., and PEARLMAN, S. J.: Hemorrhage from Pharyngeal and Peritonsillar Abscesses. *Arch. Otolaryngol.*, 18:464-509, 1933.

51. KANAVEL, A. B.: Retropharyngeal and Postmediastinal Abscesses, *Surg. Clin. N. Amer.*, 2:603-615, 1922.

52. MOORE, I.: Retropharyngeal Abscess. *Jour. Laryngol., Rhinol. and Otol.*, 33:283-284, 1918.

53. WITCHELL, I. S.: Abscess of the Brain Following Tonsillitis and Retropharyngeal Abscess. *Arch. Otolaryngol.*, 29:835-841, 1939.

54. DEAN, L. W.: The Proper Procedure for External Drainage of Retropharyngeal Abscess Secondary to Caries of the Vertebrae. *Ann. Otol., Rhinol. and Laryngol.*, 28:566-569, 1919.

55. LILLIENTHAL, H.: Retropharyngeal Abscess: The Safest Method of Drainage. *Med. Times*, 41:355-356, 1913.

56. BURCKHARDT, H.: Uber die Eroffnung der Retropharyngealen Abscesse. *Cntrlbl. f. Chir.*, p. 57, 1888.

57. BLAIR, V. P., JR.: Personal communication.

58. Dixon, O. J.: Retropharyngeal Abscess as a Cause of Respiratory Obstruction. *Jour. Kan. Med. Soc.*, 32:232-235, 1931.

59. McCoy, A. W.: Idiopathic Retropharyngeal Abscess. *Med. and Surg. Rep.* 46:313, 1882.

60. Liddell, J. A.: Abscess of the Neck. *Amer. Jour. Med. Sci.*, 85:231, 1883.

61. Davidson: Postpharyngeal Abscess. *Lancet*, 2:881, 1892.

62. Landry, M., and Billard, J.: Mastoïdite de Bezold Ouverte dans le Pharynx. *Acta Otolaryngol.*, 11:471-474, 1927.

63. Shurley, E. L.: Diseases of the Nose and Throat. New York: D. Appleton & Co., 1900, pp. 155-160.

64. Goodale, J. L.: Retropharyngeal Abscess in the Adult. *Boston Med. and Surg. Jour.*, 144:108, 1901.

65. Holmes, E. M.: Middle Ear Suppuration as an Etiologic Factor in Retropharyngeal Abscess. *Ann. Otol., Rhinol. and Laryngol.*, 15:848-867, 1906.

66. Delneuville, E.: *La Presse Otolaryngol. Belgique*, May, 1912 (quoted by Alexander and Montague, *N. Y. Med. Jour.*, 98:227-229, 1913).

67. Fulkerson, C. B.: Report of a Case of Otogenic Pharyngeal Abscess with Review of Literature. *Jour. Mich. State Med. Soc.*, 15:301-304, 1916.

68. Freudenthal, W.: In discussion of Mosher's paper.[48]

69. Babbitt, J. A.: A Study of Retropharyngeal Abscess. The Laryngoscope, 34:37-43, 1924.

70. Kelly, I. D.: Cervical Abscesses Complicating Mastoiditis. *Ann. Otol., Rhinol. and Laryngol.*, 35:23-41, 1926.

71. Glogau, O.: Abscess Descending from the Upper Air Passages into the Neck and Mediastinum. *Tran. Amer. Acad. Ophthal. and Otolaryngol.*, pp. 393-412, 1922.

72. Pratt, J. A.: In discussion of Frank's paper.[18]

73. Myers, E. L.: In discussion of Frank's paper.[18]

74. Friedman, J. and Greenfield, S. D.: Retropharyngeal Abscess: An Experience with 60 Cases. *N. Y. Med. Jour.*, 113:604-616, Apr., 1921.

75. Brooks, E. B.: Acute Retropharyngeal Abscess. *Tran. Amer. Laryngol., Rhinol. and Otol. Soc.*, 37:248-263, 1931.

76. Beyer, A. G.: Retropharyngeal Abscess Complicating a Mastoiditis in an Adult. The Laryngoscope, 41:105-107, 1931.

77. Seltz, S.: Retropharyngeal Abscess Complicating Tonsillectomy with External Approach. The Laryngoscope, 41:341-343, 1931.

78. Hempstead, B. E.: Lateral Pharyngeal Abscess Following Mastoidectomy. *Tran. Amer. Laryngol., Rhinol. and Otol. Soc.*, 39:551-553, 1933.

79. Cooper, A.: Principles and Practice of Surgery; edited by A. Lee; Vol. I, p. 79, 1836 (quoted by Mackenzie[4]).

80. Blodgett, J. H.: Quoted by Mosher.[49]

81. Evatt, C. W.: Retropharyngeal Abscess. *Jour. S. Car. Med. Assn.*, 34:287-289, Nov., 1938.

82. Bryant, F. L.: Infection of the Mouth and Pharynx. *N. Orleans Med. and Surg. Jour.*, 99:566-569, May, 1947.

83. Thorek, M., and Shapiro, M. M.: Retropharyngeal Abscess in Adults. *Jour. Internat. Coll. Surg.*, 9:671-678, Nov.-Dec., 1946.

84. Shambaugh, G. E., Jr.: Diseases of the Nose and Throat; edited by Jackson and Jackson. Philadelphia: W. B. Saunders & Co., 1945, pp. 151-152.

85. Brighton G. R.: Surgery of Nose and Throat; edited by J. D. Kernan. New York: Thos. Nelson & Sons, 1942, pp. 287-289.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of February 1, 1949.

Acousticon Model A-100.
Manufacturer: Dictograph Products Corp., 580 Fifth Ave., New York 19, N. Y.

Aurex (Semi-Portable); Aurex Model C-B, Model C-A, Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver).
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model; National Standard Model; National Star Model.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model A-1; Otarion Model A-3; Otarion Models A-4 J and S; Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear 45-CM; Radioear Model 45-M-magnetic air conduction receiver; Radioear Model 45-M-magnetic bone conduction receiver; Radioear Permo-Magnetic Uniphone; Radioear Permo-Magnetic Multipower.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Ravox (Semi-Portable).

Manufacturer: Zenith Radio Corp., 6001 W. Dickens Ave., Chicago, Ill.

Silver Micronic Hearing Aid Model 101; Silver Micronic Hearing Aid Models 202M and 202C.

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Sonotone Audicles No. 530, No. 531 and No. 533; Sonotone Model 600; Sonotone Model 700; Sonotone Model 900.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 612; Telex Model 900; Telex Model 1020; Telex Model 1550; Telex Model 1700.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Orthotronic Model; Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.

Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Radionic Model A-2-A; Zenith Radionic Model A-3-A; Zenith Radionic Model B-3-A; Zenith Model 75; Zenith Miniature 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

TABLE HEARING AIDS.

Precision Table Hearing Aid. *Jour. A. M. A.*, 139:785-786, March 19, 1949.

Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50. Report not yet published.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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